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and El Paso County Medical Society

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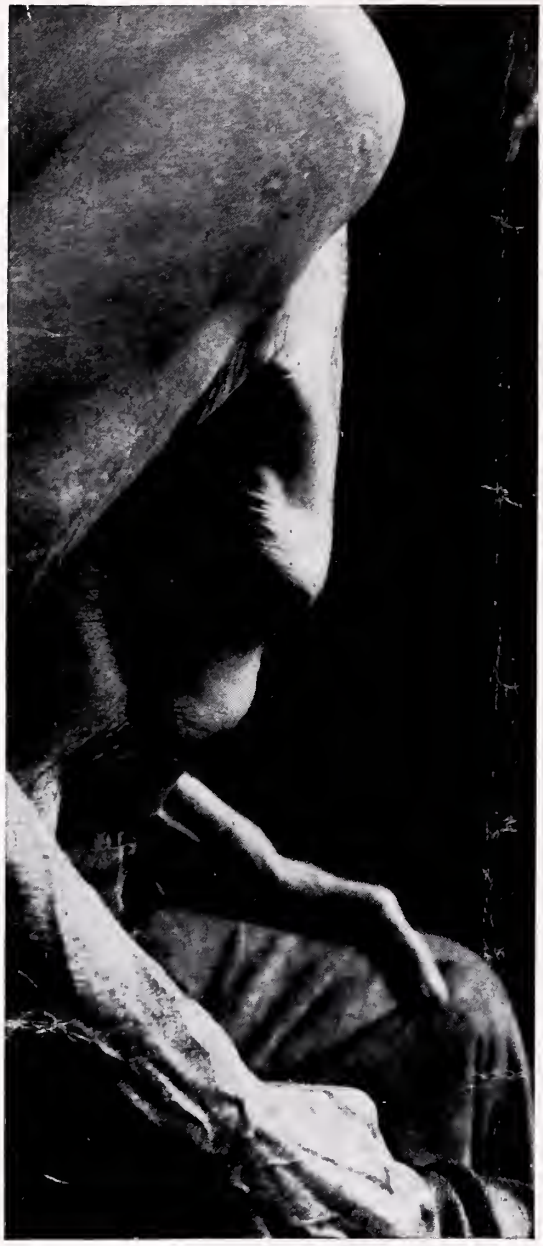

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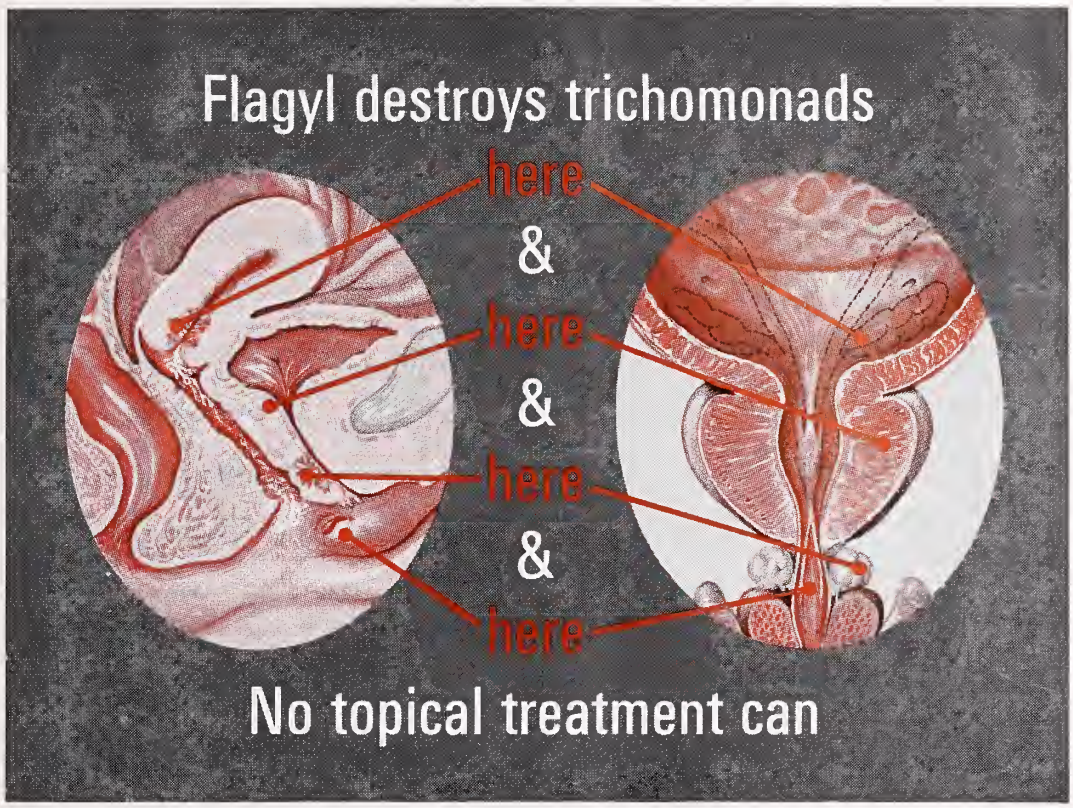

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Southwestern Medicine

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Official Journal of

The Southwestern Medical Association, The Western Association of Railway Surgeons, The Southwestern Dermatological Society, Texas District One Medical Association, The Southwestern New Mexico Medical Society, and El Paso County Medical Society

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Side effects: Side effects associated with recommended doses of 'Deprol' have been infrequent and usually easily controlled. These have included drowsiness and occasional dizziness, headache, infrequent skin rash, dryness of mouth, gastrointestinal symptoms, paresthesias, rare instances of syncope, and one case each of severe nervousness, loss of power of concentration, and withdrawal reaction (status epilepticus) after sudden discontinuation of excessive dosage.

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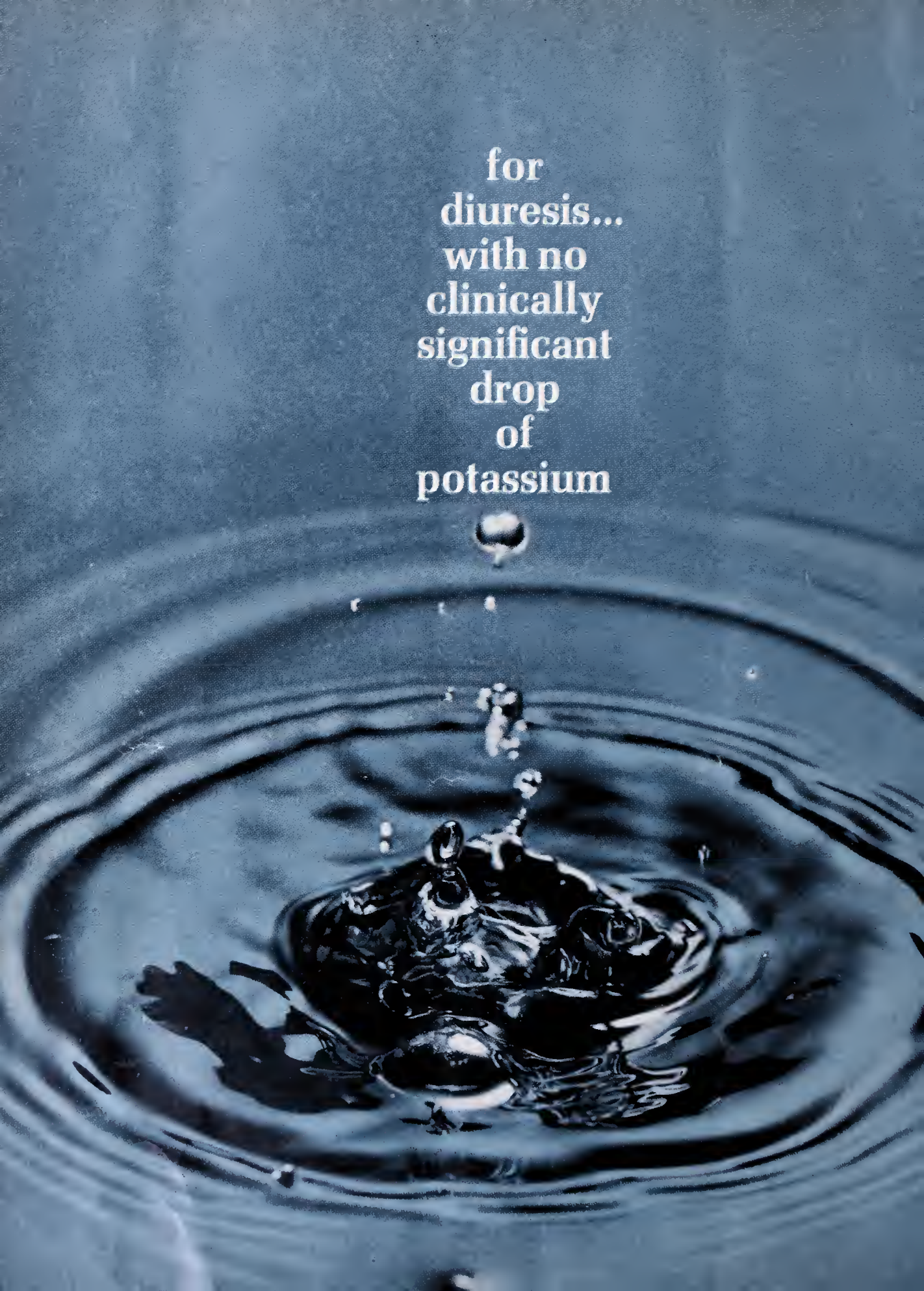
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References: 1. Swartz, C., *et al.:* Circulation 28:1042 (Dec.) 1963. 2. Published reports on file at Bristol Laboratories.

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CAUTION: This preparation may cause drowsiness. Do not drive or operate machinery while taking this medication. Do not give to children under 6 years of age or exceed recommended dose unless directed by physician. Individuals with high blood pressure, heart disease, diabetes, or thyroid disease should use only as directed by a physician. Persons with a high fever or persistent cough should not use this preparation unless directed by a physician.

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Dr. J. Travis Bennett Elected President of El Paso County Medical Society

Dr. J. Travis Bennett, a Past President of the Texas Chapter of the American Academy of Pediatrics, has been elected President of El Paso County Medical Society for the 1965-66 year. He succeeds Dr. Robert F. Boverie.

Other new officers are Dr. W. W. Schuessler, President-Elect; Dr. Erich Spier, Vice-President; Dr. Laurance N. Nickey, Secretary; Dr. B. B. Kern, Secretary-Elect; and Dr. W. L. Pierce, Treasurer.

Dr. Bennett is a Diplomate of the National Board of Medical Examiners and the American Board of Pediatrics. He is a life member of the Southern Medical Association and the American Academy of Pediatrics.

Dr. Bennett was born in Smithville, Texas, and spent his early years there and in Temple, Texas. He received his undergraduate education in the public schools of Canyon and Taylor in Texas before entering the University of Texas during World War I. At the University of Texas Dr. Bennett was a member of the Students' Army Training Corps, an Assistant in the Department of Zoology for three years, was President of the Texas Pre-Medical Society, and was initiated into Phi Beta Kappa at the end of his junior year.

After receiving a B.A. from the University of Texas, he entered Johns Hopkins University School of Medicine, from which he received his M.D. in 1926. A year's internship in Union Memorial Hospital in Baltimore was followed by two years in Pediatric training, one as Assistant Resident at the Harriet Lane Home of the Johns Hopkins Hospital and one as Resident Physician at the Hospital for Sick Children in Toronto, Canada.

Dr. Bennett spent two years in the practice of his specialty in Albuquerque before coming to El Paso in October, 1931, to become associated with Dr. Branch Craige, Sr. After Dr. Craige's death in 1944, Dr. Bennett became associated with other Pediatricians. His present associates are Dr. Ira A. Budwig, Jr., Dr. James L. McNeil and Dr. John M. Verosky. He is licensed to practice in Maryland, New Mexico and Texas.

Dr. Bennett

In addition to being a 30-year member of the Downtown Kiwanis Club, Dr. Bennett has been a Master Mason, a 32nd Degree Scottish Rite Mason and a Shriner since 1920. He has been a member of St. Clement's Episcopal Church since 1932, has served several terms on the Vestry of St. Clement's, and has served both as Junior Warden and as Senior Warden at St. Clement's. He is a member of the El Paso Chamber of Commerce, the Coranado Golf and Country Club, El Paso Museum of Art, and the University of Texas Ex-Students Association.

He is married to the former Mabel Grace Snider of Toronto, Canada. The Bennetts have one son, two daughters and four grandchildren. Their son, Travis, lives with his wife and two children in Cincinnati; their daughter, Grace Bennett Wallace lives with her husband and two children in Houston; and a daughter, Burgess Bennett, attends Texas Western College as a senior.

Some Current Concepts Concerning Accelerated Aging*

Robert E. Anderson, M.D.,** *Albuquerque*

Accelerated aging has been defined as the premature appearance of the various degenerative changes commonly associated with senility. Several experimental conditions have been shown to induce apparent accelerated aging including: chronic overfeeding; inbreeding; exposure to nonlethal amounts of ionizing radiation; increasing the environmental temperature of poikilothermic animals.[1] However, it remains unclear whether these diverse experimental conditions produce an acceleration of true "physiologic" aging or merely decreased longevity. In this connection, it is important to distinguish between aging and the pathologic alterations generally associated with senescence. Aging, in the context of this discussion, is considered to be the apparently irreversible, inevitable, apparently deleterious, temporally-related changes that result from the normal vital phenomena of an organism.[2]

The problems in attempting to evaluate the variety of experimental models which have been purported to produce accelerated aging are compounded by the large number of hypotheses relating to the etiology and pathogenesis of a physiologic aging. It is difficult to do more than speculate about the acceleration of a process that has only recently been studied experimentally. Therefore, before attempting a discussion of accelerated aging, it might be of interest to reveiw some of the currently popular concepts concerning the pathogenesis of aging.

Pathogenesis of Aging

One of the earliest concepts in this area related aging to the general "wear and tear" incurred by an organism as it attempts to adapt to its environment. This, of course, is an extension of Selye's general adaption syndrome. Every stress, therefore, produces a "scar" and the life span of an organism is related to the frequency and magnitude of the stresses and the complement of adaptive energy. When the magnitude of the stress exceeds the adaptive research, death ensues.

The somatic mutation theory relates aging to an accumulation of spontaneous, nonlethal, genetic changes which are apparently deleterious to the economy of the organism.[3]

The immunologic theory[4] is somewhat related to the above hypothesis in that aging is attributed to long-term immunologic diversification of somatic cells, possibly secondary to spontaneous mutations. Such diversification theoretically could produce immunologically "foreign" cells which then are attacked by the body's immunologic defense mechanisms. Conversely, it is possible that diversification of an immunologically competent cell could give rise to a clone capable of reacting against the body. In this connection, it is of interest that the chronic manifestations of runt disease are in many respects similar to the alterations associated with senescence.

*Presented at the Fourth Annual New Mexico Neuropsychiatric Seminar, Clovis, New Mexico, (Human Aging) New Mexico Conference for Postgraduate Education, October 1-3, 1964.

**From the University of New Mexico School of Medicine, Department of Pathology, Albuquerque, New Mexico.

Numerous theories of aging relate senescence to a variety metabolic alterations. The latter include: deterioration of cellular enzymes, perhaps occasioned by exothermic reactions; imperceptible accumulation of noxious byproducts of metabolism with depression of cellular function; malfunction of the regulatory mechanism which controls synthesis and destruction of labile molecules; alterations in the interstitial connective tissue and/or ground substance. Such a change could be due to a dysfunction of the regulatory mechanism which governs the fine balance between synthesis and destruction of labile molecules or some other alteration in the paths of communication between a cell and the cell's environment.

Radiation and Aging

Of the various experimental conditions known to induce decreased longevity (?accelerated aging), exposure to ionizing radiation has been studied most extensively and the remainder of the discussion will be confined to this area. There is no longer any doubt that there is a decrease in the life span of animals that have survived whole body irradiation and that this occurs following exposure to doses insufficient to produce early mortality.[5-7] An analysis of autopsy data reveals that irradiated animals die at an earlier age than their non-irradiated contemporaries from a variety of causes often apparently completely unrelated to acute radiation injury.[6-8] The increased incidence or accelerated appearance of select neoplasms in post-irradiation animals has long been recognized; however, the tumorigenic action of radiation does not completely explain the decreased longevity observed in post-irradiation animals.[9] Such animals show the premature appearance of a variety of degenerative changes commonly associated with senility and, therefore, are said to exhibit accelerated aging.[7]

In common with physiologic aging, the post-irradiation animals exhibit: a linear decrease in life expectancy and an increased degree or arteriolocapillary fibrosis with the passage of time, a slowly progressive generalized decrease in physiologic function involving multiple organ systems; and a temporally related increase in the incidence of malignancies. However, if radiation induces an acceleration of true physiologic aging, then it is difficult to explain why large exposures double the life span of Drosophila and small doses increase the longevity of other experimental animals.[1] Also, radiation apparently exerts a primary effect on dividing cells while the changes associated with senility have been attributed to alterations in post-mitotic cells. Obviously, further research is necessary to resolve these apparent inconsistencies.

Accelerated aging following irradiation has only been studied to a very limited degree in humans and quantitation has been very difficult. Warren has demonstrated decreased longevity among American radiologists[10] but similar changes have not been confirmed among their British counterparts.[11] Previous studies performed among the survivors of the Hiroshima atomic bombing of August 6, 1945 have not shown evidence of accelerated aging but generally have not employed a quantifiable parameter. As has been pointed out by Sobel,[12] in order to demonstrate that radiation accelerates true "physiologic" aging, and does not just nonspecifically reduce longevity, it is necessary to: 1) define a quantifiable biochemical or physiological phenomenon which is age dependent, 2) show that changes in this age-dependent phenomenon are accelerated post-irradiation.

The rate of mucopolysaccharide ground substance, including hexosamine, to collagen in a variety of tissues (skin, lung, femur, sternum, aorta, and inferior vena cava) has been shown to be an age-related phenomenon in rats, guinea pigs, rabbits and man,[13,14] and this ratio has been generally accepted as a measure of biochemical age.[12-18] Therefore, application of this parameter to survivors of the atomic bomb is of some interest.

Hexosamine and collagen determinations were performed on skin and aorta obtained from men autopsied at the Atomic Bomb Casualty Commission during the period 1962-1964. Cases were grouped by age without knowledge of the clinical or autopsy findings as follows: tissue from a proximally exposed individual (less than 1400 meters from the hypocenter at the time of the explosion) was paired with tissue from an individual not in Hiroshima at the time of the bomb. Only tissue from proximally exposed survivors was employed since there is general acceptance that these persons absorbed a biologically significant amount of radiation at the time of the bomb.

Chemical determinations were performed on tissue obtained from 14 experimental pairs (28 individuals). In 11 of the pairs the hexosamine: collagen ratio of the skin and aorta was smaller

in the exposed member than the nonexposed one.[2] The probability of obtaining such a distribution by chance sampling is statistically unlikely ($p<0.05$).

These biochemical alterations, demonstrated in persons with no other known morphologic, clinical or chemical indication of previous exposure to significant amounts of ionizing radiation, strongly suggest the possibility of accelerated aging among proximally exposed survivors of the atomic bomb. This conclusion is only valid, however, if the hexosamine: collagen ratio represents a true parameter of aging. Although this appears to be a valid assumption at this time, the field of experimental gerontology is sufficiently immature that new information often requires re-evaluation of accepted premises and data. Therefore, the apparent association between aging and radiation in humans should be considered as tentative and reinterpreted in the light of new information concerning accelerated and physiologic aging.

Summary

This report reviews some of the experimental data and currently popular concepts concerning the pathogenesis of aging and relates these hypotheses to accelerated aging and especially the form which is apparently associated with sublethal amounts of ionizing radiation.

References

1. Walford, R.L.: Further Considerations Towards an Immunologic Theory of Aging. Exp. Geront. 1:67, 1964.
2. Anderson, R.E.: Aging in Hiroshima Atomic Bomb Survivors. Arch. Path. 79:1, 1965.
3. Failla, G: The Aging Process and Somatic Mutations: In: The Biology of Aging, a Symposium. Am. Inst. Biol. Sci., Washington, D.C., Publ. Co., p. 170.
4. Walford, R.L.: Auto-immunity and Aging. J. Geront. 17:281. 1962.
5. Alexander, P.: Accelerated Aging: Long-term Effects of Exposure to Ionizing Radiations, Gerontologia (Basel) 1:174, 1957.
6. Lamson, B.G., et al: Late Effects of Total-body Roentgen Irradiations: III. Early appearance of neoplasms and life-shortening in female Wistar rats surviving 1,000 r hypoxic total-body irradiation, AMA Arch. Path. 66:311, 1958.
7. Upton, A.C.: Ionizing Radiation and Aging Process, J. Geront. 12:306, 1957.
8. Upton, A.C., Furth, J., and Christenberry, K.W.: Late Effects of Thermal Neutron Irradiation in Mice, Cancer Res. 14:682, 1954.
9. Alexander, P., and Connell, D.J.: Shortening of Life Span of Mice by Irradiation with X-rays and Treatment with Radiomimetic Chemicals, Radiat. Res. 12:38, 1960.
10. Warren, S.: Longevity and Causes of Death from Irradiation in Physicians, JAMA 162:464, 1956.
11. Court-Brown, W.M., and Doll, R.: Expectation of Life and Mortality from Cancer Among British Radiologists, Brit. Med. J. 2:181, 1958.
12. Sobel, H., Gabay, S., and Bonorris, G.: Studies on Hexosamine and Collagen in Tissues of Rats Which Survived X-irradiation, J. Geront. 15:253, 1960.
13. Sobel, H., and Marmorston, J.: Possible Role of Gel-fiber Ratio of Connective Tissue in Aging Process, J. Geront. 11:2, 1956.
14. Sobel, H., et al: Influence of Age Upon Hexosamine-collagen Ratio of Dermal Biopsies from Men, J. Geront. 13:128, 1958.
15. Sobel, H., Marmorston, J., and Moore, F.: Collagen and Hexosamine Content of Femurs of Rats, Proc. Soc. Exp. Biol. Med. 87:346, 1954.
16. Sobel, H., Zutrauen, H.A., and Marmorston, J.: Collagen and Hexosamine Content of Skin of Normal and Experimentally Treated Rats, Arch. Biochem. 46:221, 1953.
17. Clausen, B.: Influence of Age on Connective Tissue: Hexosamine and Hydroxyproline in Human Aorta, Myocardium and Skin, Lab. Invest. 11:229, 1962.
18. Kao, K.T., Hilker, D.M., and McGavack, T.H.: Connective Tissue: III. Collagen and Hexosamine Content of Tissues of Rats of Different Ages, Proc. Soc. Exp. Biol. Med. 104: 359, 1960.

N. M. Society to Discuss Medicare

Recent legislation passed by Congress involving the medical profession will be discussed at the Annual Conference of County Medical Society Officers throughout the State in Santa Fe, January 15, 1966.

The meeting is designed to provide membership of the New Mexico Medical Society with the most recent, up-to-date information available on this legislation, according to Dr. Omar Legant, Albuquerque, Chairman of the State Society's Public Relations Committee, which has planned the program.

Among speakers will be Bernard D. Hirsch, LL.B., Director of the American Medical Association Law Department; Dr. Russell B. Roth, member of the AMA Advisory Committee on physician participation on Medicare; Mrs. Richard A. Sutter, President of the AMA Auxiliary; Dr. Edwin O. Wicks, Director of the New Mexico Department of Public Health; L. W. Burrell, Executive Director of New Mexico Blue Cross-Blue Shield; Leo Murphy, Director of the New Mexico Department of Public Welfare; and Dr. Reginald Fitz, Dean of the University of New Mexico School of Medicine.

Subjects to be discussed include regional medical centers, voluntary area-wide hospital planning, hospital utilization committees, physicians' responsibility under Medicare, and compensation to physicians under the usual and customary fee concept.

Dr. Robert P. Beaudette, Raton, is President of the State Society.

Headquarters for the meeting will be the La Fonda.

Cranberry Juice In The Treatment Of Urinary Tract Infections

Prodromos N. Papas, M.D.,* Charles A. Brusch, M.D.,**
and George C. Ceresia, Ph.D.***

Infections of the urinary tract, a basic problem in urology, frequently confront almost every practitioner of medicine. It is commonly accepted that they are second in incidence only to viral and bacterial infections of the upper respiratory tract. The most prevalent renal lesion found at necropsy is chronic pyelonephritis. Even when rigid histologic criteria are applied the incidence of active or healed pyelonephritis in consecutive hospital autopsies is between three per cent and six per cent.[17] The evidence from these post-mortem studies is that a surprisingly high percentage of chronic pyelonephritis is undiagnosed in life.[12] While the interrelationship between acute urinary tract infection and chronic pyelonephritis is still not clearly understood, some workers have questioned the cause-and-effect relationship between symptomatic or asymptomatic bacterial infection of the urinary tract and later chronic pyelonephritis.[17] However, the available evidence[7] supports the hypothesis that persistent or recurrent bacterial infection can lead to the development of pyelonephritis. Population surveys among girls[23] and women[14] have revealed the prevalence of unsuspected and asymptomatic infection of the urinary tract and have shown that such a condition among pregnant women can progress to pyelonephritis. Asymptomatic infection of the urinary tract can and often does continue[16,18] from one pregnancy to the next, becoming more potentially dangerous with each succeeding pregnancy.[4,8,20,27]

While the pathogenesis, natural history, and diagnosis of pyelonephritis continue to be investigated it seems rational to assume that all urinary tract infection is potentially harmful and could progress to chronic pyelonephritis and its possible sequelae, hypertension and renal failure. The earlier and more completely acute infections of the urinary tract are eradicated the better is the ultimate renal prognosis. It would appear germane to surmise that urinary infection, including renal involvement, is likely to occur in patients having a significant degree of persistent bacteriuris.[19] Patients such as pregnant women and diabetics who are especially susceptible to asymptomatic bacteriuria should have routine examination of the urine to enable early detection and therapy.

For many years cranberry juice has been used for the management of urinary tract infections. Data concerning the cranberry juice's reputed symptomatic relief of urinary tract infections has been limited. In 1914 it was demonstrated that most of the various organic acids present in various fruits are completely oxidized within the body and do not exhibit any acid effect in urine. However, the acids present in cranberries, prunes and plums proved the exception.[1] In that same year it was reported that cranberries contained 0.06

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per cent benzoic acid.[30] Nine years later it was found that 24 hours after the ingestion of 305 gm. of cranberries marked increases, in both titratable and organic acids, and a decrease in pH of the urine were produced. The increased acidity of the urine was attributed to the synthesis and excretion of hippuric acid. Since the amount of benzoic acid ingested was far too small to serve as an intermediate compound for the large amounts of hippuric acid found in the urine it was postulated that quinic acid was the principal source of the hippuric acid.[2] This hypothesis was later substantiated[29] and in 1931 quinic acid was isolated from cranberries.[21] In 1933 it was shown that the ingestion of 100-300 gm. of cranberries increased the titratable acidity, organic acids, hippuric acid, hydrogen ion concentration and ammonia while the uric acid and urea nitrogen of urine were decreased slightly. Hippuric acid gave the best determination of the quantity of cranberries ingested since the amount found in the urine was directly proportional to the weight of cranberries eaten.[9] More recently it was reported that the ingestion of large quantities of cranberry juice increased the hippuric acid content of urine by several grams a day.[3] This increase in hippuric acid excretion was accompanied by small decreases in urine pH. In vivo tests established that 0.02-0.04 M of hippuric acid was bacteriostatic at pH 5.0 for common pathogens of the urinary tract but this action was considerably decreased as the urine pH was raised. Test results of six normal male subjects showed that at times cranberry juice contributed enough hippuric acid to the urine to achieve concentrations which were bacteriostatic at pH 5.0. Twelve ounces of cranberry juice per day relieved urinary symptoms in a group of patients with chronic urethritis, with or without caruncle formation, and those with so-called trigonitis.[26] The antibacterial effect of extended cranberry juice therapy was discussed in 1964.[35] At this time it was stated that the antibacterial effect of cranberry juice was considerably enhanced when used in conjunction with methenamine. In that same year a study of the effects of cranberry juice on ammoniacal fermentation in voided urine was performed at two institutions. The daily ingestion of 16 ounces of cranberry juice resulted in the abatement of odor of ammoniacal fermentation in voided urine. The results suggested that cranberry juice was apparently bacteriostatic enough to inhibit ammoniacal fermentation.[22] The present study was undertaken to investigate the effects of cranberry juice in the treatment of acute infections of the urinary tract.

Materials and Methods

The subjects of this investigation were 60 adults who presented definite symptoms of acute urinary tract infections such as frequency, dysuria, urgency, nocturia. Of the 60 patients, 44 were females. Thirty of the women were under 40 years of age: 11 of the males were over 40. Twenty-six of the patients had some prior history of urinary tract infection. All patients were without overt evidence of underlying uropathy or predisposing disease.

Catheterization is one of the age-old procedures for obtaining urine specimens aseptically from females, and this method continues to be preferred by some urologists. Within the past decade, however, investigators[14,15,26,31] have shown that clean-voided, midstream urine specimens from patients with significant bacteriuria usually contains 100,000 or more organisms per ml. Conversely, contaminents will rarely be present in concentrations greater than 10,000 per ml. Today, many clinicians believe that since such quantitative culture of the urine appears to be quite reliable and, since the microbiologic correlation between specimens obtained by the clean-voided technique and by catheterization from the same individual exceeds 95 per cent, catheterization for microbiologic diagnosis is unnecessary. The clean-voided technique[24] also avoids physical, psychologic and infectious hazards.[5,11,10,28,33] To limit outside contamination of the urine specimen obtained via the clean-voided technique, it is axiomatic that care must be taken in the cleansing of the genitalia.[13]

Urine specimens were obtained under the following conditions. In males the glans penis was cleansed with aqueous zephiran solution or 1:1000 merthiolate followed with hydrogen peroxide. A clean-voided, second-glass specimen was then obtained. In females the external genitalia was carefully cleaned with soap and water then with either merthiolate or aqueous zephiran followed by hydrogen peroxide. The labia were separated and a clean-voided, second-glass specimen procurred. Urine specimens were taken directly to the laboratory for culture.

The method of quantitation used was a pour-plate culture, in which 1 ml. of each of a 1:100 and a 1:1000 dilution of urine was flooded with

cooled, melted agar. After incubation for 48 hours at 37 C, the plates were counted. The presence of 100,000 or more bacteria, cultured from 1 ml. of urine specimens collected aseptically by the clean-voided technique, has been established for cases having a significant bacteriuria. If clean-voided, midstream specimens are processed without delay from non-infected cases, the cultures rarely demonstrate numbers which approach this critical level.[13,14,25] We have used this criteria in this study for the detection of true bacteriuria. However, we do recognize that there are disadvantages in too much emphasis on an absolute figure and that the significance of the bacteriological count should be assessed in terms of its relation to clinically apparent urinary infection.[32,34] In addition to urine cultures, white blood cell and differential counts and routine urinalysis were performed. No patient was being treated with antibacterial medication for urinary tract infection when admitted to the study. All patients ingested 16 ounces of cranberry juice per day for the 21-day therapy period. The juice used in this study was the commercial cranberry product. Post-treatment, clean-voided urine specimens were aseptically collected and cultured at the cessation of therapy and again after six weeks.

Results

Pre-therapy urine culture in 22 (37 per cent) of 60 patients failed to show a significant bacteriuria based upon the criteria of the presence of 100,000 or more bacteria per ml. of urine. Urine cultures of four of these 22 patients showed no growth at all and were considered sterile. Frequency of micturition and dysuria were just as common in those patients that did not have a significant bacteriuria as in those that did. All of the 38 infected patients had an excess of white cells in the urine. Ten of the patients presenting with symptoms but with less than 100,000 bacteria per ml. of urine also had an excess of white cells. The organisms cultured included the whole spectrum of common urinary tract pathogens. Gram-negative bacilli and gram-positive cocci predominated, the ratio of the former to the latter being approximately 6:1. Escherichia coli followed by Proteus, Pseudomonas and Klebsiella were the most prevalent. Infection due to multiple organisms was present in seven (12%) patients.

After three weeks of cranberry juice therapy a positive clinical response, (no urogential complaints and fewer than 100,000 bacteria per ml. of urine) was noted in 32 (53%) patients. An additional 12 (20%) patients were judged to be moderately improved. Sixteen (27%) patients showed neither bacteriological improvement nor symptomatic relief. Clinically the cranberry juice was well accepted by the patients and there were no clinical side effects. Follow-up on the 44 cases in which clinical and bacteriological improvement was observed was undertaken six weeks after termination of therapy. Infection persisted or recurred during the six weeks after treatment in 27 (61%) of the 44 cases. Symptomatic relief was noted in eight of these cases. Seventeen (39%) patients had negative urine cultures and remained free of clinical complaints. One of the four patients with sterile pre-therapy urine developed asymptomatic bacteriuria.

Discussion

It is now known that the activity of drugs in the management of urinary infections is enhanced by the adjustment of urinary pH.[6] The tetracyclines, cycloserine, and novobiocin appear to be more potent at acid pH. Methenamine mandelate has been widely used for suppressing bacterial multiplication. The mandelic acid component of methenamine mandelate aids in acidification of the urine releasing formaldehyde from methenamine. The formaldehyde is antibacterial to both gram-negative and gram-positive organisms. Methenamine mandelate's antibacterial activity, however, falls off sharply as urinary pH rises. Since cranberry juice lowers urine pH and increases the hippuric acid content of urine, it would appear that it has definite merits as an inexpensive, nontoxic and palatable acidifying agent with food value. When used in conjunction with methenamine its antibacterial effect is considerably increased. Its use in long-term suppressive therapy in combination with other bacteriostatic and bactericidal agents is indicated. Long-term acidification has been shown to be most promising in children with repeated bouts of bacterial infection in the urine.

We feel that the substantial decrease in bacterial count and the alleviation of urogenital complaint after three weeks of cranberry juice therapy is of significance. Exceptional patient tolerance to the juice was noted. Even in those cases where complete elimination of the pathogens was not achieved, clinical improvement was shown. Al-

though critical evaluation of the long-term bacteriological results did not indicate a permanent inhibition of the bacterial count, the short-term suppression of bacterial flora and clinical symptoms was gratifying. Since acute infections of the urinary tract are most common, the use of a nontoxic, nonantibiotic bacteriostatic agent such as cranberry juice has a continued place in urogenital therapeutics.

Summary

Cranberry juice was administered to 60 patients with clinical diagnoses of acute infection of the urinary tract. After three weeks of treatment with 16 ounces of cranberry juice per day, 53 per cent of the patients had a positive clinical response. Moderate improvement was noted in an additional 20 per cent of the patients. Exceptional patient tolerance to the cranberry juice was noted. Infection persisted or recurred during the six weeks after treatment in 27 of the patients. Eight of these 27 patients were asymptomatic. Negative urine cultures and absence of clinical complaints were noted in 17 patients at the six week post-therapy follow-up.

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References

1. Blatherwick,N.R.: Arch. Int. Med. 14:409,1914.
2. Blatherwick,N.R. and Long,M.L.: J. Biol. Chem. 57:815,1923.
3. Bodel,P.T., Cotran,R., and Kass,E.H.: J. of Lab. & Clin. Med. 54:881,1959.
4. Boswell,R.R., and MacLoren,E.: S. G. & O. 114:583,1962.
5. Brumfitt,W., Davis,B.L., and Rosser,E.: Lancet. 2:1059,1961.
6. Brumfitt,W., and Percival,A.: Lancet, 1:186,1962.
7. Brumfitt,W., and Percival,A.: J. Clin. Path. 17:482,1964.
8. Bulkeley,G.: Med. Clinic of N. Amer. 45:1,1961.
9. Fellers,C.R., Redman,B.C., and Parrot,E.M.: J. Nutrition. 6:455,1933.
10. Guze,L.B., and Beeson,P.B.: New Eng. J. Med. 255:474,1956.
11. Hinkle,Nancy, Partin,J.C., and West,C.D.: Amer. J. Dis. Chil. 100:333,1960.
12. Jackson,G.G., Dallenbach,F.D., and Kipnis,G.P.: Med. Clin. N. Amer. 39:297,1955.
13. Kaitz,A.L., and Williams,E.J.: New Engl. J. Med. 262:425,1960.
14. Kass,E.H.: Trans. Assn. Amer. Phys. 69:56,1956.
15. Kass,E.H.: Arch. Inter. Med. 100:709,1957.
16. Kass,E.H.: Arch. Int. Med. 105:194,1960.
17. Kimmelstiel,P., Kim,O.J., Beres,J.A., and Wellman,K.: Amer. J. Med. 30:589,1961.
18. Kincaid-Smith,P., and Bullen,M.: Lancet 1:395,1965.
19. Kleeman,C.R., Hewitt,W.L., and Guze,L.B.: Medicine 39:3. 1960.
20. Klein,L.: Am. J. Obs.-Gyn. 83:528,1962.
21. Kohman,E.F., and Sanborn,N.H.: J. Ind. Eng. Chem. 23:126, 1931.
22. Kraemer,R.J.: Southwestern Med. 45:7,1964.
23. Kunin,C.M., Deutscher,R., and Paquin,A.: Medicine (Baltimore), 43:91,1964.
24. Little,P.J.: Lancet. 1:1149,1962.
25. Merritt,A.D., and Stanford,J.P.: J. Lab. Clin. Med. 52:463. 1958.
26. Moen,D.V.: Wisconsin Med. J. 61:282,1962.
27. Monzon,O.T., Ory,E.M., Dobson,H.L., Carter,E., and Yow, E.M.: New Engl. J. Med. 259:764,1948.
28. Monzon,O.T., Armstrong,D., Pion,R.J., Deigh, Rosalyn, and Hewitt,W.L.: Amer. J. Obs.-Gyn. 85:511,1963.
29. Pryles,C.V., and Steg,Nina L.: Pediatrics 23:441,1959.
30. Quick,A.J.: J. Biol. Chem. 92:65,1931.
31. Radin,M.J.: J. Ind. & Eng. Chem. 6:518,1914.
32. Riley,H.D.,Jr.: J. Lab. Clin. Med. 52:840,1958.
33. Seneca,H., and Peer,P.: J. Urol. 95:78,1965.
34. Turner,A.C.: Lancet 2:1062,1961.
35. Yow,E.M., Monzon,O.T., Ory,E.M., and Brennan,J.C.: In: Biology of Pyelonephritis. London: J&A Churchill. Ltd., 1960.
36. Zinsser,H.H.: Med. Clin. N. Amer. 48:293,1964.

TMA District One Meets in El Paso, Feb. 5

Dr. David Wade, Austin, President of the Texas Medical Association, will be the banquet speaker Feb. 5 in El Paso at a one-day joint meeting of District One of the TMA and the El Paso County Medical Society.

The program will start in the Hilton Inn with a luncheon at 12:30 p.m. A business meeting will be held at 1:15 p.m. and the scientific program will run from 2 to 4 p.m. Cocktails, to be followed by the banquet, will start at 6:30 p.m.

Scientific presentations will be as follows: "A Mathematical—Electronic Approach to the Diagnosis of Certain Internal Derangements of the Knee Joint" by Dr. Louis W. Breck, El Paso; "Secondary Operations for So-Called Post Cholescystectomy Syndrome" by Dr. Victor M. Blanco, El Paso; "Recent Developments in the Management of Head Injuries" by Dr. K. Zolfoghary, El Paso; and "Urinary Tract Infections in Children" by Dr. Jose Roman, Jr., El Paso.

Officers of District One are Dr. Ira A. Budwig, Jr., El Paso, President; Dr. Cecil Robinson, Kermit, President-Elect; Dr. Mario Palafox, El Paso, Secretary-Treasurer; and Dr. Russell Holt, Councilor.

Mrs. Roy F. Kemper, Coleman, Texas, Regional Vice-President of the TMA Auxiliary, will speak at a luncheon for wives at noon in Ardovino's Restaurant. Mrs. Jesson L. Stowe, El Paso, is in charge of the Auxiliary program.

Letter to the Editor

In the August issue of SOUTHWESTERN MEDICINE one of my fellow graduates of the University of Texas, a noble institution steeped in the legends of the past with such pre-eminent physicians as William Keiler, James Thompson, A. O. Singleton and a host of others, appealed for us to "accept the Challenge," "that we fit in as a necessity in any society," "that we can survive no matter what," *etc.*

The monument on Bunker Hill is not a monument to a British victory. It stands as a symbol of the end of British tyranny and the beginning of American freedom. The torch was lighted by American patriotism and adherence to principle.

I would ask my colleagues to consider carefully the fees paid the physician in Hungary, or Poland, or the U. S. S. R., or any of the countries where the Orwellian prophecy of 1984 arrived some years past with a paean of totally triumphant socialism. It was George Washington who said, "government is like fire — a wonderful servant but a dreadful master." There is no difference in the totalitarian concept regardless of the wishes of the physician caught up in the maelstrom he would rather "ride out" simply attending to patients and "leaving politics to others." The fee for apathy and indifference is terror and slavery. Today the menacles and leg-irons are being forged for patient and physician alike and, "when freedom fails, what remains?"

As physicians we ignored the collectivist trends on all sides until our own professional status was threatened and then found ourselves in the corner without an exit. We can repeat that tactical maneuver of waiting, as our so-called 'leaders' have asked, until the "chips are down" and repeat an exercise in futility evident at another Texas shrine — the Alamo. As God Almighty was their witness, not one of those brave men who fought for the right with backs to the wall ever sought a compromise with evil or thought of walking as slaves back into the Dark Ages. And yet our worthy colleague sings a siren song of ignoring evil and simply attending the sick while Doctor Ernesto "Che" Guevara and other collectivists "doctor sick nations."

True, we have a responsibility to the sick and yet the evidence points overwhelmingly to the superiority of medicine and our allied professions, as we have known it and them these many years — some longer than most — but imbued with the ideals and knowledge that ours was and is the finest system in the world. Examine the new drug discoveries of the past 23 years and not a single one has come from the "collectivist camp."

This today is our testing time. Yours and yours alone is the decision. Yet the pages of that great book will be inscribed with it and the effect upon those billions enslaved and as yet unborn. The greatest Christian theologian of all time expressed an attitude for those of us who are by virtue of education, temperament and training more nobly suited for dissent. Paul said in his Second Letter to the Corinthians, "Be ye not unequally yoked together for what company hath righteousness with unrighteousness and what communion hath light with darkness?"

Shall we sit out a wake as long and as dark and dreary as this one for the American Republic and the peoples of the world while we consider fees of potatoes and quarters of beef?

Forbid it, Almighty God!

George S. Richardson, M.D.
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Coming Meetings

27th Annual Southwest Allergy Forum, Plaza Motor Hotel, El Paso, Jan. 20-22, 1966.

Texas Medical Association's Conference on Medical Service and Legislation, TMA Headquarters, Austin, January 22, 1966.

District I meeting of the TMA, Hilton Inn, El Paso, Feb. 5, 1966.

1965-66 Psychiatric Seminar, sponsored by the New Mexico Medical Society, New Mexico Chapter, AAGP, New Mexico State Hospital, University of New Mexico School of Medicine, State Hospital, Las Vegas, New Mexico, March 4-5, 1966.

99th Annual Session of the Texas Medical Association, Austin, April 14-17, 1966.

84th Annual Meeting of the New Mexico Medical Society, Western Skies Hotel, Albuquerque, May 10-13, 1966. Business Meetings—Western Skies—May 10-11, Clinical Programs—Student Union Building, University of New Mexico, May 12-13.

9th Annual Ruidoso Summer Clinic sponsored by the New Mexico Chapter of the American Academy of General Practice, Ruidoso, N.M., Chaparral Motel, July 25-28, 1966.

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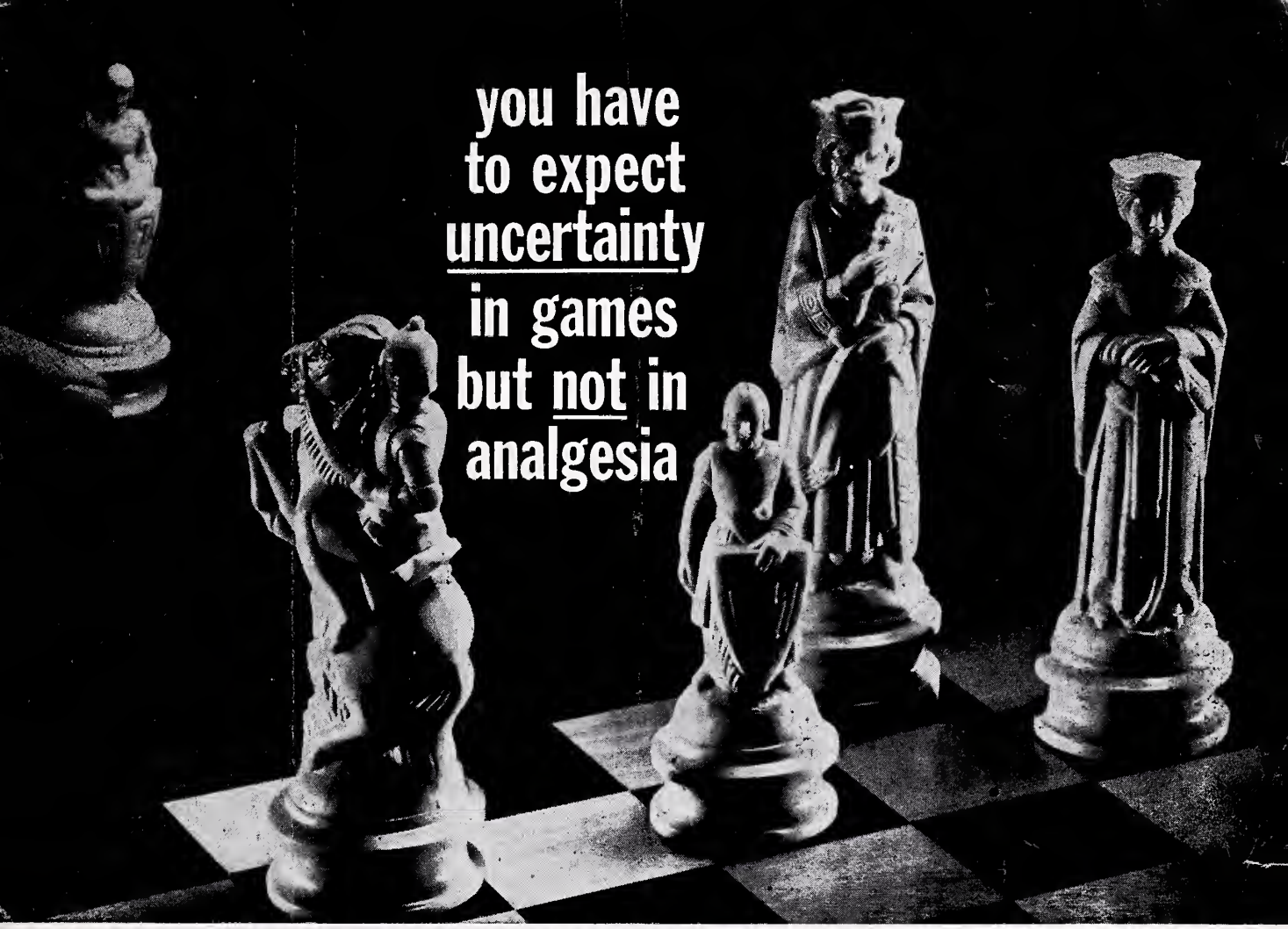

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Southwestern MEDICINE

Official Journal of the Southwestern Medical Association,
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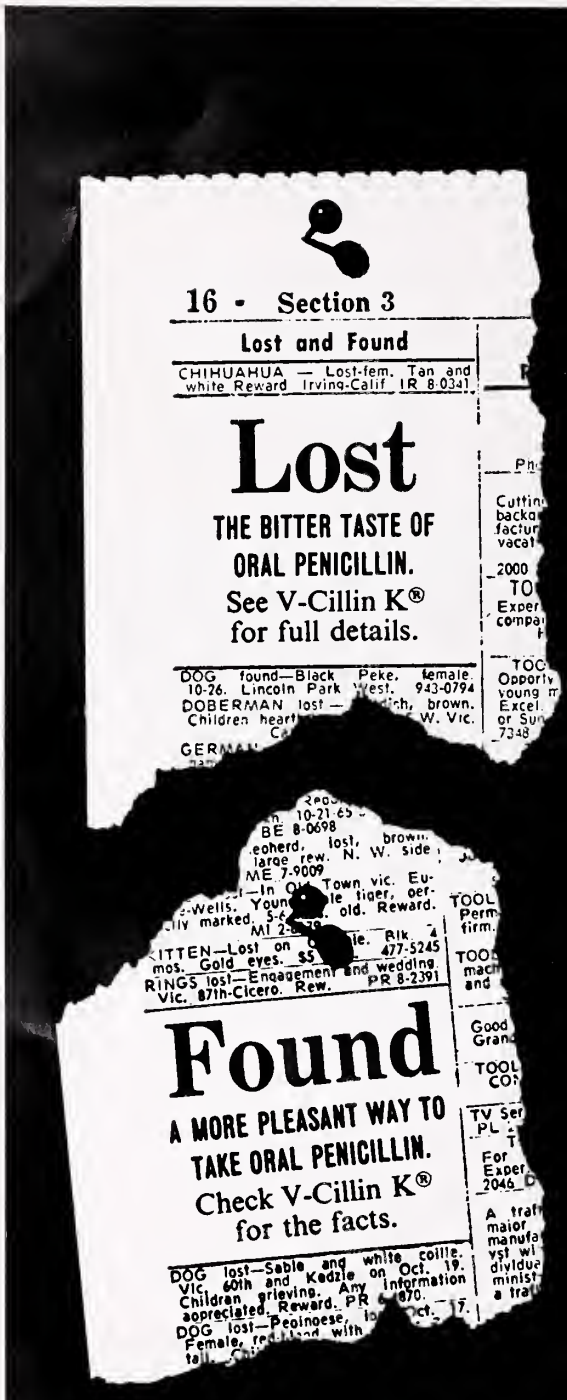
and El Paso County Medical Society

IN THIS ISSUE

VOL. 47, NO. 2

February, 1966

Founded 1916



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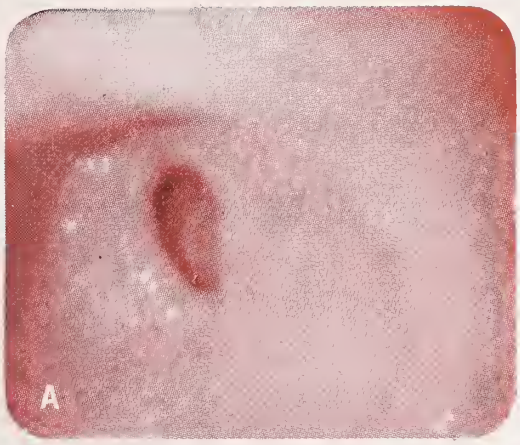
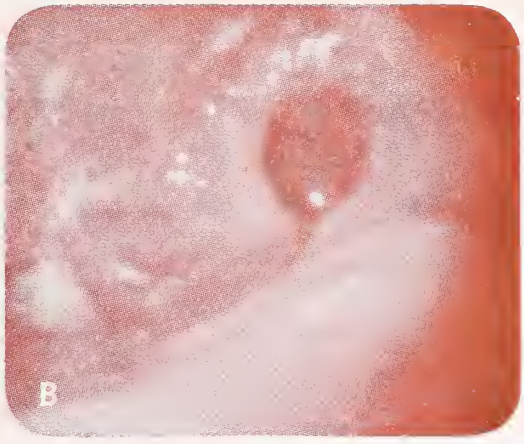


Which Is Pyloroplasty with Vagotomy?



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Photographs—Harry Barowsky, M.D., Lawrence Greene, M.D., and Robert Bennett, M.D., from a Scientific Exhibit presented at the Annual Meeting of the American College of Gastroenterology, Bar Harbour, Florida, Oct. 24-27, 1965.

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Southwestern Medicine

The U. S.-Mexico Regional Medical Journal Serving West Texas, New Mexico, Arizona, Nevada and Northern Mexico

Official Journal of

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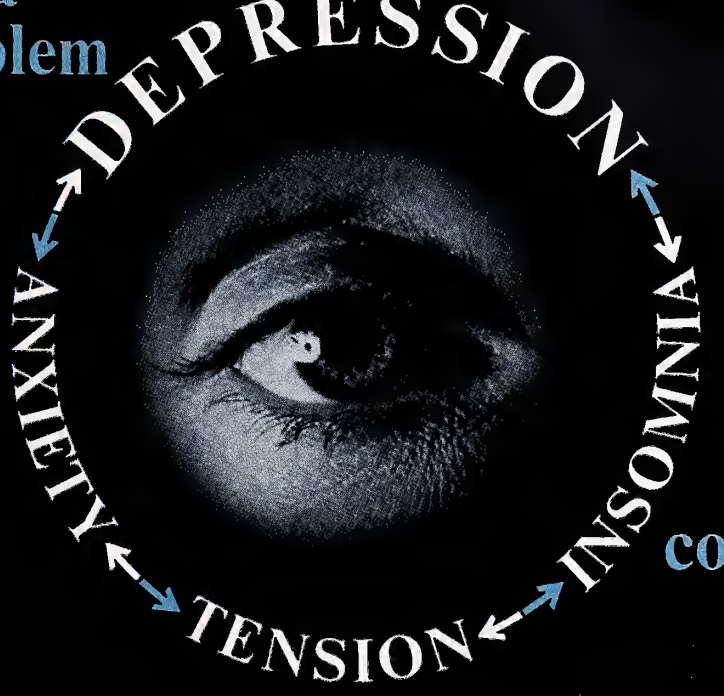

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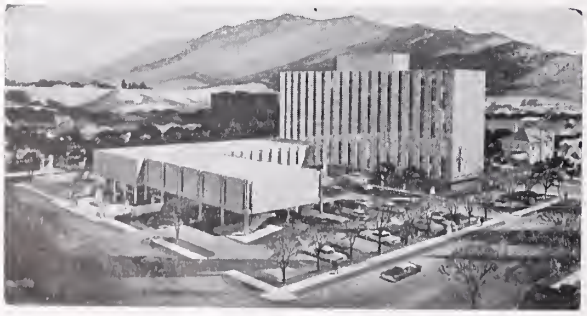

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14th Annual Scott and White Conference in Medicine and Surgery, Scott and White Clinic, Temple, Texas, Feb. 20-22, 1966.

31st Midwinter Clinical Session of the Colorado Medical Society, Brown Palace Hotel, Denver, March 1-3, 1966.

1965-66 Psychiatric Seminar, sponsored by the New Mexico Medical Society, New Mexico Chapter, AAGP, New Mexico State Hospital, University of New Mexico School of Medicine, State Hospital, Las Vegas, New Mexico, March 4-5, 1966.

20th Annual Symposium on Fundamental Cancer Research, University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, March 7-9, 1966.

Third Teratology Workshop, University of Colorado, Boulder, April 4-8, 1966.

99th Annual Session of the Texas Medical Association, Austin, April 14-17, 1966.

84th Annual Meeting of the New Mexico Medical Society, Western Skies Hotel, Albuquerque, May 10-13, 1966. Business Meetings—Western Skies—May 10-11, Clinical Programs—Student Union Building, University of New Mexico, May 12-13.

Fourth National Instrument Society of America (ISA) Symposium on Biomedical Sciences Instrumentation, Disneyland Hotel, Anaheim, California, May 16-19, 1966.

9th Annual Ruidoso Summer Clinic sponsored by the New Mexico Chapter of the American Academy of General Practice, Ruidoso, N.M., Chaparral Motel, July 25-28, 1966.

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"Eat, Herbie, eat. Don't you want to grow up big and strong?"

Man In Space

Lt. Col. Robert H. Moser,* MC, *El Paso*

(The opinions expressed in this article are solely those of the author and do not reflect the policies or attitudes of the National Aeronautics and Space Administration or the U.S. Army.)

Within the decade, a man will walk on the surface of the moon. Whether he will plant an American or a Soviet flag is a matter of vast political significance. However, when the ultimate history of mankind is written the critical fact recorded will be, that in this year, man succeeded in wrenching free of the gravitational bindings of our planet to traverse the untraversable, land safely on the moon and return to earth. This will represent a momentous intermediate step in interplanetary travel beyond the moon.

The integration of man and space vehicle represents an experiment of limitless imagination and technicological skill. In the early days when interplanetary exploration was still an exercise in mathematical probability and bright young space engineers were exchanging practical dreams around large tables, there was a significant angry whisper concerning the role of man in the early exploration of space. Engineers are a practical breed. They deal in a world of mathematical accuracy where performance is predictable, error is reprehensible and indecision unknown. Many felt that magical little black boxes could gather data concerning the mysteries of space that would be reliable and informative, without the necessity of integrating human frailty into the mathematical equation. There is much to be said for this philosophy. To date we have launched over 100 successful unmanned space vehicles — the remarkable success of the Tiros weather satellites, the Pioneer V Venus probe, Mariner IV Mars probe, Syncom, OGO, the Telestar communications satellites, and multiple others attest to this.

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The average astronaut weights 160 pounds, occupies approximately six cubic feet and has a built-in potential for error. The engineer suspected that he could reduplicate virtually everything that man could do, with black boxes of less weight and mass. In addition, the integration of man into the capsule introduced many other problems.

Man is a most delicate creature. To protect him during launch and re-entry and support his viability in the hostile environment of space presented an enormous task. Investigation of man's tolerance of space was to be added to the early data-gathering mission of the spacecraft.

Finally the discussion was resolved. The engineers were convinced that despite the abhorrent weight and mass of man it was perhaps worth the effort — because man had a brain. The little black boxes could come pretty close but even the clever engineers could not program a computer that could *think* with elasticity and *initiate action* and still compress it into as small a package as the human brain. Despite several tons of sophisticated, unemotional hardware whirling about this sphere — each firing its lovely orderly data to busy earth-bound computers — only John Glenn saw the "fireflies." Human eyes are required to appreciate the ethereal beauty of space. In a more practical sense, manual re-entries have been flown in Mercury and Gemini when the black boxes faltered. Perhaps I have been somewhat facetious, but I can assure you that until the successful flights of Carpenter, Grissom and Glenn there were many of us who were concerned as to whether man had been integrated into the spacecraft prematurely.

In a more practical way let us discuss the problems that beset our engineering friends. For the

moment let us speak of the relatively — and I stress relatively — simple problem of orbital flight, remembering at all times that the problems of deep space travel will be far more complex. The Titan booster and the Gemini capsule represent the ultimate in American engineering sophistication. How far we have come in the integration of the man into the spacecraft is revealed by a brief look at the orientation of the Mercury-Gemini programs.

Our sole purpose was to prove and improve man's capability to survive the vicissitudes of launch, survival in near space, and re-entry. Prior to the moment when Alan Shephard was sealed into Freedom 7 atop a Redstone ballistic missile, seven highly trained, supremely motivated and physically superior young men were subjected to every type of stress that could be anticipated during an orbital space voyage with the exception of the prolonged weightless state. In the Mercury program, from knowledge gained during ballistic flight of Atlas missiles, it was ascertained that the maximum G load to which astronauts would be subjected during nominal flight, would be in the range of seven G's. In the event of an abort situation in which the astronaut was hurled from the top of an ailing Atlas booster by firing of the escape rocket, a maximum load of 15-20 G's for a fraction of a second was anticipated. The seat ejection mechanism of Gemini is somewhat more arduous. It was also estimated that in the event of a land impact the astronaut would be subjected to a force of approximately 30-40 gravities for a split second. This prompted investigations on the rocket sled at Holloman Air Force Base in which anesthetized animals were subjected to 38 times the force of gravity for short impulses. Up to 20 second exposure at this G load was within the range of tolerance. Human subjects have taken transverse decelerations of 40 G's for 0.5 seconds without injury. It was estimated that a human subject within a capsule in the restrained, aft facing, semisupine tranverse orientation could survive an 80 G knot impact on land. In addition, all astronauts were dispatched to the Human Centrifuge at Johnsville, Pennsylvania. On this incredible device they were accelerated up to 14 G's positive gravity for brief periods of time. Most of them were able to tolerate this quite well with actual improvement in performance with repeated exposure. Actually several were subjected to a nine G turnover which means that they were accelerated to nine G's positive pressure ("eyeballs in") and then suddenly flipped 180° to nine G's negative pressure ("eyeballs out"). This also was tolerated without blackout. Thus, the astronauts were well prepared for the G-loading forces to which they would be subjected in launch and re-entry on Mercury and Gemini missions.

Noise and vibration were considered potential sources of hazard. Tapes were recorded at various distances and positions from booster engines during static firing. These were played to the astronauts during periods while they were solving cockpit problems under conditions of simulated flight. The loudest sound on the inside of an Atlas borne capsule has been recorded at 165 decibels; Titan is similar. This intensity is well attenuated before reaching the pilot's ear drums.

The only vibration difficulty encountered in actual flight was in the early launch phases of the Redstone mission when the missiles were approaching "max Q" where the forces of velocity and the resistance of gravity and atmospheric density grapple, to produce maximum stress on missile, spacecraft, and man. Both Shephard and Grissom observed some vibration. It was not a problem on Atlas missions. Cooper and Conrad experienced significant "POGO effect" — a vertical oscillation during launch of GT-5.

A major consideration in design of the life support system was oxygen supply and CO_2 removal. This has not been a problem. For about two hours prior to launch, the suit and later, the cabin environment is flushed with 100 per cent oxygen which the astronauts breathe; waste gases are vented out of the spacecraft. This continues up to a few seconds before the umbilical cable separates from the capsule and the on-board O_2 cuts in. During this period approximately 97 per cent of body nitrogen will be washed out. Thereafter, the astronauts fly in a 100 per cent oxygen environment maintained at an atmospheric pressure of about 5.1 pounds per square inch, which is equivalent to 259 mm of mercury or 27,000 feet altitude. Throughout the flight, 100 per cent oxygen is delivered to the crew from a system containing gaseous oxygen under high pressure which goes through a series of reducing valves

to deliver it to the suit inlet. I won't go into the details of the many safety features built into this system. Suffice it to say that the suit with face-plate closed and the cabin have completely independent environmental control systems. In Gemini each suit is autonomous. This is important re-duplication. In the rare event of cabin perforation by a meteorite or an electrical fire or a gaseous leak requiring the cabin gas contents to be dumped overboard, the astronauts may close the face-plate and live in the suit environment in safety and comfort for at least two revolutions. In GT-5 Cooper and Conrad flew with helmet and gloves off except during launch and re-entry. The crew of GT-7 flew in their underwear.

Carbon dioxide retention has not presented a significant problem. This gas is absorbed by a lithium hydroxide system which takes up carbon dioxide as it is vented from the suit outlet. Detection of rising pCO_2 has represented somewhat of a problem. Instruments sufficiently sensitive to detect the rather infinitesimal amounts of pCO_2 that may make the difference between tolerable limits and hypercapnic toxicity are now available. This was a problem in Mercury. For example, nominal pCO_2 at the suit inlet is around .02 p.s.i. or 1.0 mm of mercury. Earliest toxic signs and symptoms begin at .05 p.s.i. or 2.6 mm of mercury and reach distinctly toxic levels at 10 mm of mercury. By comparison, with oxygen we are dealing with larger magnitudes, ranges of 5.1 p.s.i. or 259 mm of mercury *optimally*. Danger areas begin at 3.8 p.s.i. or 197 mm of mercury. We also rely on clinical signs of hypercapnia; increases in depth and rate of respiration and pilot comfort and orientation are end-points. This has not been a problem to date.

Pressure within the cabin, as I have stated, is maintained at 259 mm of mercury. Since oxygen is the only gas in the system, cabin pressure is equal to O_2 partial pressure. This greatly simplifies matters.

Heat was considered a major environmental hazard in early planning. The engineers assured us the problem was solved and indeed it is. Once the retro rockets have been fired and the spacecraft has been slowed to suborbital velocity, it begins its searing descent through the ever-thickening atmosphere. From 55 miles altitude to 12 miles, which represents a slant distance of about 760 miles, the spacecraft will decelerate from 17,500 miles an hour to about 270 miles an hour. This occupies a terribly long five minutes. It is during this period that the maximum heat build-up occurs. With the spacecraft oriented at -1.5°, the heat shield is deployed to accept the friction; there is a maximum heat pulse of about 3,000F at the apex of the ablation shield for about two minutes. This heat deflector consists of a tough fiberglass resin which flakes off during re-entry. The temperature transmitted to the rest of the spacecraft is considerably decreased. The outer wall of the beryllium shingled cabin peaks at about 1000° in the area of the cockpit. The inner cabin gas temperature may rise to about 170°. However, during the orbital missions thus far, this has actually been in the range of $90\text{-}106^{\circ}$ in Mercury and less during Gemini. The temperature within the suit, with face-plate closed, has been in the area of $66\text{-}86^{\circ}$ and the body temperature of the astronauts has not changed at all. We think this is pretty good refrigeration.

I don't want to go into all the details of environmental and life-support systems on re-entry. Suffice it to say, they have all worked with great effectiveness. The astronauts are exposed to the atmosphere of earth at approximately 27,000 feet through the automatic opening of snorkel valves. Cabin temperatures on the water have actually been a greater problem than those encountered during re-entry. This is primarily due to the high temperature and humidity in the waters off the Florida coast. If ambient temperature exceeds 97° or humidity 58 per cent, the astronauts are obliged to leave the capsule. You are familiar with the problem of orthostatic hypotension experienced by Gordon Cooper after the 22 orbits of MA-9. I am delighted to report that although there was some laboratory evidence of loss of cardiovascular "tone," this has not been a problem in any of the Gemini flights. Mild demineralization was anticipated and has been detected by sensitive radiographic techniques; both the cardiovascular and demineralization are rapidly reversible.

Looking to the future, we have many new and different problems. It is evident that man living in the closed ecologic system of the spacecraft at O G for periods of several weeks to months may present a new constellation of problems. In all probability man must fly with an atmospheric environment that simulates the natural state on

earth. Certainly the eight days of GT-5 were well tolerated. However there is theoretical reason to believe that breathing 100 per cent oxygen for more prolonged periods of time, even at reduced pressure, may be deleterious. Significant work has been going on for years in the area of the "flying farm," where a closed and balanced system utilizing algae in the production of oxygen and the utilization of CO_2 has been studied. Storage and preservation of food and disposal or utilization of waste will be problems. One can discuss such things as cosmic radiation, ecplexia (the psychologic phenomenon of break-off as one watches the earth disappear in the darkness of space), boredom, isolation, fatigue and, not the least, the mystery of prolonged weightlessness. Soon manned orbiting laboratories will extend experience and permit studies of physiologic reactions for prolonged space voyages. In past lectures I have said one cannot expect that the human organism, which for thousands of years of painful evolution has adapted its homeostatic mechanisms to the upright posture of one G, will not experience some physiologic disruption when deprived of this attitude for prolonged periods of time. But I must revise this. The incredible capability of the human organism to adapt to this new environment has exceeded our most optimistic expectations. We have been exploring new techniques for replacing gravitational sensations which may keep our proprioceptors happy.

In conclusion, may I say that we are in reach of the moon and beyond. Man is an active participant in this exciting adventure. The disciplines of medicine and astronautics have been married in this greatest of scientific ventures. Our horizons are (literally) unlimited.

The Family Doctor—The Aged Patient—And Family Therapy

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One of my favorite stories about Sir William Osler — the father image for all of us — concerns the time when in his later years he became ill and was taken to the hospital. He was, of course, attended by a very large team of devoted physicians, a team that was undoubtedly very concerned for this wonderful man, but, on the other hand, probably had differences of opinions and wanted to try different courses of treatment. The upshot of the story is that Sir William survived and left the hospital, nicely recovered from his illness. When the nurse pulled the bed back from the wall, she noticed that there were a number of little bumps in the wallpaper. Upon investigating the source of these bumps, it was found that these were pills that Sir William had secretly stuffed under the wallpaper. A further check of the whole situation revealed that under that lumpy wallpaper were all the pills that had been prescribed for Sir William during his hospital stay. The team of physicians who cared for him must have indeed thought that he was an irascible, senile old man who was becoming difficult to treat — which may have been true — but he recovered. He may have shown a great deal of wisdom in his own right. So it is with the older person; they can never be underestimated as to their powers to recover from illness or the surprisingly wise things they sometimes do. One of the things we have to constantly guard against is underselling these attributes in the older, aged individual.

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Family Therapy

Psychiatrists fall into this trap no less than others. Several years ago, a new technique became a "fad" in psychiatry. This was the so-called development of "family therapy." The concept of this approach is to help patients with emotional problems and involve the entire family in a group

therapy process. The individual who first sought help was seen as mirroring the psychopathology in the family itself. The entire family would be brought in for initial interviews and evaluation sessions, and then if significant problems were found to exist, the entire family was enrolled in "family therapy." Different people throughout the country had varying degrees of success with this procedure, but it soon became apparent to several investigators that one of the reasons for their failures was the fact that they had overlooked "grandma" or "grandad" or an older aunt or relative who was living in the home at the time. They had been casually passed by — overlooked — because it was thought that these older people "would not benefit from psychotherapy." This sort of thinking was based on the therapist's previous experience that the older person often does not benefit from the conventional forms of psychotherapy in the same way that the middle-aged or younger person does. Of course, the thing they overlooked was the fact that family group therapy is indeed a unique and different approach to psychotherapy.

In this approach to solving the emotional ills of people in a given family, our present ground rules are as follows: Everyone in the family is seen in a series of initial evaluation interviews. My own preference is in the first interview to see everybody living in the immediate household. This includes even small children and, of course, any older people who may be living in the home. After the first interview, the younger children, generally those under age 12, are dropped from further interviews, although we do make exceptions to this. The reason for including even babies in the initial interview is to observe how different members of the family relate to each other. It is interesting, for instance, to see if father or an older uncle seems to pay any attention to the small children at all, or perhaps pays too much attention. The behavioral relationships of the family as a whole are carefully observed in the first interview as well as any verbal interchanges than can be elicited.

An Example

An example of the sort of material one can get from the family interview technique is illustrated by the following clinical example from my own practice. Recently, I had the opportunity of interviewing a family consisting of the following members:

(1) The mother who came in because of her own symptoms of severe depression and anxiety. At times, her anxiety made her seem almost inappropriate and initially I wondered if she might not be psychotic. She was a most unhappy and distressed person, but in the first interview — alone with me — she could come up with no reason for this. She had a nice home, a nice family and a "perfect" husband.

(2) The second member was the husband who was a successful rancher, who through the years had obtained large holdings in livestock and had built a very nice "spread" for himself and his family. He was also a leader in the community and a pillar of his church.

(3) This couple had four children, the oldest a boy 16 who was in some rebellion with the family though in a quiet and modest sort of way. He was described mostly as a hardworking, quiet young man who would on rare occasions do things such as steal hub caps.

(4) A 12-year-old daughter who was given to fits of "temper" but by and large was described as a very quiet, cooperative young lady who helped her mother a great deal.

(5) The third child was six years old, a boy, and was still enuretic three or four nights per week. The mother also described a speech problem in this youngster, a "stammering" when under any sort of pressure. He had indicated a fairly severe school phobia when he started the first grade some months before, but this had been largely overcome, according to the mother.

(6) The fourth child was a three-year-old boy who was also brought to the initial family interview.

(7) Living in the ranch home also was a 72-year-old grandfather, the wife's father. The wife in her initial interview described him as a quiet but lovable old man and stated that the children adored him and that he made a special point of keeping out of her husband's road (even at that she was afraid at times her husband might have resented him). The grandfather lived six months with this particular family and then six months with the wife's sister's family in a nearby community. This initial interview was held behind a one-way vision screen with residents and medical students watching as I conducted the interview.

Family Interview

The first family interview was most interesting and revealing. It was a warm spring day, and this

particular examining room was located two stories above street level and all of the windows were open with one of the windows being unscreened. The father came in and took the largest "easy" chair. The mother with all of the children closely huddled about her, sat on a couch. Grandad sat quietly in an opposite corner of the room from the father. After the purpose of the interview was explained to the entire family, the father was the first to become active in the interview. He expressed his doubts that such an interview was necessary, explained that his wife was the "sick one" and that the rest of the family was free of any emotional disturbance — except perhaps for the 16-year-old boy who would be all right if he would just "shape up." I encouraged the 16-year-old boy to talk and for the first time in his life, under the protective environment of the interview situation, he was able to speak directly with his father and to tell him of some of the ways that he resented his attitude of authority. The father quickly denied that he was at all authoritarian and preferred to paint himself as the lovable father image who controlled the family not by demands and rules, but rather by good example. About this time, the mother let the three-year-old down on the floor and the baby began to explore the various attractive parts of the interview room. The grandfather, the mother, and so far all of the children, except for the 16-year-old, had been very quiet and guarded in this initial interview.

About halfway through the interview, the three-year-old toddler seemed to be working himself dangerously near the open, unscreened window. I was becoming uneasy about his closeness to the window, but the interesting thing was that the family was doing nothing about it. I watched for several minutes and as he got still closer to the window, I was on the verge of going over and picking him up and taking him back to a safer place in the room. The medical students and residents watching behind the one-way vision mirror were becoming uneasy and afterwards reported to me they were about to rap on the window because they thought I had not noticed what the child was doing. The entire family was well aware of the child's closeness to the window; they were watching the child closely. The interview conversation had become very quiet. None of the family members made any move to do anything about the child. The grandfather was sitting closest to the child. Next was the mother and then the other children on the couch. I was across the room on one side. The father was across the room on another side and actually was the farthest from the child. The father had just finished describing how he thought his family was a real democracy and that as the leader of the democracy, certainly he was not a dictator in any sense of the word. At the point where my uneasiness had about led me to the decision to go over and bring the child back to a safer spot in the room, the father suddenly jumped from his chair and in a few quick strides across the room, pulled off his heavy leather belt and before the startled eyes of all of us (including those behind the one-way vision mirror), the belt snapped out and cracked the three-year-old a resounding blow. The father then picked up the three-year-old and literally threw him in the mother's lap, put his belt back on, sat back in his chair as if to continue where he left off. The family, as it turned out in later interviews, had expected this exact thing to happen. "We wouldn't dare ever do anything — that is Dad's job," was a later "family" opinion.

This initial interview was followed by several other evaluation interviews, and the family was eventually treated by the process of family therapy. I'll digress from the description of the family for a moment to tell you the sorts of things we look for in such an interview situation and then use this family as illustrative examples of the points I am going to make.

In the initial family interviews, we are basically looking for three things: (1) Who has the symptom? (2) Who has the pathology? (This is often *not* the person who has the symptom.) (3) What member or members are capable of making healthy changes that could alter the family psychopathology?

Now if we look back at the family I have described, mother obviously had the symptoms. Her depression and anxiety made her so uncomfortable that she sought help for herself initially. In spite of the fact that she was the one who was "hurting," she was completely unable in my first session with her to describe the sort of family interaction I saw during the first family interview. She was unable to talk about the cause of the distressful and depressing family interactions, namely, the severe tyrannical, authoritarian role played by the

husband. After the family interview, we could label the husband as having the major pathology. It was also our feeling that because of his severe rigidity and the complete way he had dominated all family members for so many years, he was the least likely to show any initial change. As we looked for the third point, namely, who was most apt to show change that would relieve this pathological family interaction, we looked to the 16-year-old boy and the grandfather. We felt that perhaps these two together could make some changes that would alter the family problems. Unlike the mother, these two realized the tremendous struggle with the authoritarian father, but up to this point they had been unable to do anything about it.

Father Involved

After several family interviews which served to point out more vividly the problems I have already described, it was decided to take the family into therapy and to see father, mother, grandfather, the 16-year-old and 12-year-old child on a once-a-week basis. The group met for an hour and 15 minutes once a week. The observers behind the one-way screen soon labeled the father as the "fastest belt in the West," and, as it developed, the family was obviously very cowed, subdued and frightened of this belt, whether it was the actual belt itself or the "belting" attitude taken by the father towards the entire aspect of family living. As this pathology became apparent to the family, the situation became very threatening to the father. *Often in family therapy the crucial factor will be whether father will or will not stay in therapy because in a sense he loses his place in the family to the therapist.* He is in some degree replaced by the therapist; this alone can be quite threatening and was especially so in this situation where the father was so authoritarian. However, because the entire family is involved in the therapeutic process, it is also very difficult for the father to quit therapy. The rest of the family is working at it, and if he quits, he then appears to be the one who "runs."

In this particular instance, the father withdrew to a haughty position and remained aloof and disdaining of the whole family interaction as it took place in the therapeutic setting. The evolution of the family was that grandfather was able to see his place in helping the 16-year-old develop some healthy independence of his tyrannical father. The grandfather and the 16-year-old were able together to get involved in projects and relationships that were not dominated by the father. After the 16-year-old was helped in some of these by his grandfather, he was then able to reach out on his own and for the first time became a little independent from the severe authoritarian figure in the family. Grandfather's life became a great deal happier too, because he found himself useful now in helping his grandchildren cope with the situation which he had seen for many years but which he had always felt was "none of my business" and out of the realm of his ability to change. The therapist was able to use the grandfather's love and understanding to point out to the father that this sort of leadership is generally more effective than the "belt." The mother was able to follow the 16-year-old and gain at least some measure of independence so that most of her depressive symptoms lifted. All of this was not easy on the father. He would withdraw and sulk during the sessions and obviously was quite angry at the therapist. As the family moved into a healthier adjustment of independence, and the mother's depression had lifted to the point where she was relatively comfortable, the father was then convinced that he should enter into individual psychotherapy. This was necessary before the family really reached the sort of emotional adjustment that was necessary for them to remain relatively symptom-free.

Who Has What

This case represents the sort of situation one finds where the oldster in the family is counted on to make healthy changes. This may be a little surprising because we often think of the oldster as being the one who either has the symptoms and/or the pathology. Of course, there are times when the aged patient may have the pathology or may have the symptoms—and sometimes even both—but the family interview technique is an efficient and, I think, relatively rapid way of finding out "who has what."

Probably it is common in your practices to encounter a situation where the oldster has the problem and the family itself has the symptoms. I am referring to the older person who develops certain senile changes. He may become hard to manage, become in some ways a behavioral problem, and perhaps be inclined to wander about and get lost. He may become very forgetful and have all of the

other symptoms that can develop with advancing years. The family then develops the symptoms because of their concern and worry over the oldster. They may become anxious and upset about him in many ways, and emotional conflicts within the family itself may develop. A family interview may not be required in such a circumstance because the report of family members or perhaps examination of the oldster reveals him as the one with the pathology. It is often amazing what good medical care can do to alleviate some of the senile changes. The doctor can check to be sure nutritional needs are met and that environmental circumstances are such that the oldster can be realistically expected to handle them. In other words, being sure that the oldster has good medical care will often bring about substantial changes in the problem, so that symptoms in the family disappear.

Senile Changes

There are times that even with the best of management and the closest attention paid to nutritional and metabolic balances, the person of advanced years will still show signs of senile changes. If one of these signs (particularly if it is a disturbing influence to the family) is agitated behavior, perhaps some tranquilizer may be useful. In using the tranquilizing drugs for agitated behavior, one must recognize that the drug is used exactly for that. The drugs control behavior or change behavior so that the patient can then be helped in other ways. This is true not only with the older patient but with any patient for which the tranquilizing drugs are prescribed. An example is the schizophrenic patient. The drug itself has no real effect on the schizophrenic process, but merely is given to the patient to control some undesired or frightening sort of behavior, so that the patient may then be helped with other sorts of therapy techniques. Sometimes, of course, we are all called upon to recommend a nursing home situation or an environment where the oldster can be better cared for than in the home. When this is required, the removal of the problem, in this case the oldster, may alleviate family symptoms. However, one has to be careful it doesn't produce new symptoms, i.e., those of guilt over not taking care of the older person, or the development of conflict among family members themselves because of disagreement over the recommendation for moving the oldster to a nursing home. Here the physician can at times take the responsibility for the decision because it is often easier for the family to deal with a medical decision and recommendation rather than to accept the opinion of one of the other family members.

You probably have also seen the situation where the oldester has the symptoms but the family really has the problem. In this instance the older person is living in a home where there is a great deal of family conflict and turmoil. He may actually become a scapegoat for this. Because of his advancing years and his position within the family he is unable to see his rightful place in this conflict and so may be the brunt of a lot of emotional abuse, for which he is really not responsible. In these cases I believe these families really should be involved in family group therapy. They have to come to some settlement and adjustment of the family conflicts themselves before they can ever handle the oldster in a comfortable manner. In such a circumstance, again, the physician may be in the position of making a medical recommendation to change the environment of the older person to a nursing home, perhaps on a temporary basis, until the family comes to some basic resolution of its own conflicts. Even in these situations we find it helpful to bring the older person into the family group sessions at times so that he can understand the family's problems, especially if he is expected in the future to return to the family home.

In the above instance, the oldster may be the one who is initially seen in the office because he is the one with the symptoms. These symptoms may range from depression to inappropriate behavior because of the tremendous frustration he feels.

Summary

We have gone over material representing three different ways in which the person of advancing years may find himself involved in family pathology:

(1) He may actually be the problem itself and give rise to symptoms in the family. Here the treatment is focussed on the oldster himself.

(2) He may have the symptoms and not be the problem. Here the family conflict is not the oldster and is beyond his control. The family needs treatment to relieve the aged patient of his symptoms.

(3) The oldster may be the one in the family who can be expected to make healthy and perhaps fairly rapid changes. This is probably the most important point we have learned from "Family Therapy."

The Child With The Persistent Cough

(Cystic Fibrosis)

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Introduction

A child coughs because there is irritation in his respiratory system. This may be in the upper respiratory system encompassing the throat, or it may be down in the lower respiratory system encompassing the lungs. In coughing, he attempts to clear the respiratory passageways of irritation, whether it be due to infection, excessive secretions or an irritating factor which may be inhaled.

Common causes of persistent cough in the child are whooping cough, asthma, allergies, tuberculosis, chronic bronchitis, pneumonia, postnasal drip, and irritation from smoke and dust. One of the most serious and most common of the life-threatening chronic pulmonary diseases of childhood is cystic fibrosis which kills more children than any other disease of childhood except cancer. We consider this as probably the worst pulmonary respiratory condition of childhood.

Incidence and Etiology

Cystic fibrosis is not a contagious disease. It cannot be transferred from one child to the other; rather, it is inherited. Every two and a half hours a cystic fibrosis child is born today in the United States, or about one in every thousand babies. Seven thousand cases are diagnosed annually, making an estimated 25,000 living American children and young adults with the disease. Many remain undiagnosed. The cause is thought to be a Mendelian recessive genetic trait, inherited from both parents, each of whom must be a carrier of the recessive gene. One in every 20 individuals in the country are carriers of the potential producing gene. The odds of two such persons marrying is one in 400. One out of four of their children will inherit the cystic fibrosis gene from both parents and be a cystic fibrosis patient; two may inherit the gene from only one parent and be carriers, and one child may be normal. Both sexes are affected equally. The age of the parents has no influence on the incidence, nor does the birth order of the child.

What Is Cystic Fibrosis?

Cystic fibrosis is believed to be caused by a lack of or insufficiency of some vital chemical substance, possibly an enzyme or hormone, which is essential to normal functioning of the sweat, mucus and other glands of external secretion. This basic biochemical defect is produced by the abnormal gene, and remains unknown today. Normally, the mucus secreted by the lining membranes of the organs is thin, clear and slippery. Mucus produced in the cystic fibrosis patient is thick, and sticky and may obstruct the intestines, block the ducts of the pancreas and liver and plug up the air passageways in the lungs.

Fanconi, a Swiss pediatrician, reported in 1936 two children with pancreatic insufficiency together with pulmonary disease and suggested the name of cystic fibrosis of the pancreas. Dr. Dorothy Anderson of New York City described the complete pathology in the pancreatic glands in 1938. The thick, tenacious mucus obstructs the small ducts of the pancreas which supply important enzymes to the small intestine for the digestion of fats, proteins and carbohydrates. As these small ducts in the pancreas become obstructed by this thick, abnormal secretion, cysts and scarring develop which result in the cystic fibrosis picture. Deprived of sufficient enzymes from the pancreas, the child is unable to digest his food properly. The liver and other digestive glands may also have the tiny ducts clogged by this abnormal mucus, causing the secretory cells of the liver to die, and become replaced by a fat and scar tissue.

Among the non-mucus producing glands of the exocrine system which are affected are the sweat glands, the tear glands and the salivary glands. In

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the sweat glands, the absence or deficiency of some unknown substance in the body tissue produces malfunctioning of the sweat glands, resulting in excessive sweating plus an inability to control the amount of salt in the sweat. This may lead to heat prostration. Likewise, there is an increase in the chlorides or salt in the tears. In the salivary glands there is an inability to control the loss of salt in the secretions, resulting in a higher salt content than in the normal salivary secretion.

In the lungs the abnormal mucus obstructs the air ducts, creating conditions which make the lungs susceptible to infection and leading to chronic bronchitis, bronchopneumonia, and emphysema, with air-trapping, leading to the barrel-type chest.

Clinical Manifestations

Clinical manifestations can also be divided according to the various systems affected. Many times in infancy there may be intestinal obstruction, producing what is called "Meconium ileus" (This occurs in about 10 per cent of all infants born with this disease). In others, during infancy, there may be a voracious appetite with weight loss, distension of the abdomen, very little subcutaneous tissue with thin buttocks, quite large stools which are foamy and foul smelling, and rectal prolapse may occur.

As far as the sweat and other electrolyte systems are concerned, the sweat is many times more salty than the normal; there may be heat prostration and the salivary glands may be quite enlarged.

Pulmonary symptoms consist of fits of coughing, frequently quite prolonged, which may cause vomiting and many times interfere with sleep. The lack of oxygen weakens the child; he may have rapid and shallow breathing; it is difficult to get enough oxygen for even the smallest physical effort, and he, therefore, is unable to run and play with other children and frequently must be confined indoors. In addition to a chest configuration, resulting in a pigeon-like chest, there may be a bluish color to his lips and nails due to cyanosis; and clubbing of the fingers and toes due to inadequate circulation to the fingers and toes. Frequently these children are of small stature, nonathletic, but they are usually quite intelligent and realistic; they are quite mature emotionally; they have to face the facts of life and early death.

Diagnosis

Cystic fibrosis may often be confused with chronic diarrhea, celiac disease, malnutrition, or with asthma, allergies, emphysema, tuberculosis, chronic bronchitis, pneumonia and whooping cough. The diagnosis is usually established by (1) observation of the clinical findings, (2) X-Ray findings and (3) laboratory tests. A positive family history helps. There is no single symptom or laboratory test that can absolutely determine the diagnosis.

In addition to the clinical manifestations mentioned above, X-Ray findings frequently show emphysema with air trapping and chronic bronchopneumonic areas with scarring.

Among the laboratory tests which are available for absolute, positive diagnosis are (1) collection of pancreatic enzymes with a determination of deficiency, (2) the sweat test, and a test of the salivary juices which usually show an increase in chlorides. The easiest and most positive diagnostic test available today is the sweat test which can be done on a rapid screening basis, by a palm test. Here the hand is placed on a silver nitrate medium in a glass dish. With an increase in the concentration of salt in the sweat, the more yellow the hand imprint becomes. A more accurate sweat test is done, however, by iontophoresis. Here a sweat-stimulating chemical is injected into the forearm of the child, a small pad is placed over this and the child is stimulated to sweat. This is collected over a period of 45 to 60 minutes and the sweat is then analyzed from this test. Any sweat chloride over 60 Meg/L is considered as diagnostic.

Treatment

Treatment is a 24 hour a day proposition both for the patient and the parents. There are three important therapy areas: 1. Pulmonary Obstruction 2. Secondary Pulmonary Infection 3. Correction of pancreatic deficiency and control of the nutritional aspects. Suppose we take the last of these first. It is necessary to control the diet by giving a high protein, low fat, moderate carbohydrate type of diet, adding pancreatic extracts to the diet so that the foods may be properly digested. Also large doses of vitamins are given as there may be some vitamin deficiency present. Lastly, one must insure adequate water and salt intake.

About the treatment of the pulmonary aspects—first one must treat the pulmonary infection and get this under control with the use of antibiotic, given by mouth, or frequently by injection or by

aerosol — breathing these in, or a combination of methods of administration. The obstruction must be treated by nebulization, by having these children sleep in a mist tent, which produces a fine fog which will keep their passageways loosened up and clear. Children must be taught postural drainage and breathing exercises; their physical activities must be controlled. Many times medicines to promote expectoration of sputum are given, together with drugs which will dilate the bronchial or air passageways. Environmental factors are of significant importance in that one should avoid dust and other irritants, as well as significant changes in temperature and humidity.

Frequently children may require hospitalization. Home care and therapy require a cautious and continuous follow-up by a team well acquainted with the disease process.

Prognosis and the Future

As more becomes known of cystic fibrosis, the life expectancy increases beyond infancy and childhood to the possibility of young adulthood and even further. The prognosis depends a great deal on the control of pulmonary infection and disease. There is no known cure for cystic fibrosis at this time, but research and education are leading us to new and better methods of care. Possibly some day the cause will be determined and then the cure can be effected.

Until such time, it is wise for all parents of children with a persistent cough to call this to the attention of their physician so that these children may be investigated for the most serious of all respiratory conditions in children — cystic fibrosis.

Psychiatric Seminar in Las Vegas, N. M., March 4-5

The Fifth Annual Psychiatric Seminar, sponsored by the New Mexico Medical Society, the New Mexico Chapter of the AAGP, the New Mexico State Hospital, and the New Mexico University School of Medicine, will be held March 4 and 5, 1966, at the State Hospital in Las Vegas, New Mexico. The Seminar is designed primarily for physicians who are not Psychiatrists.

Guest speakers will be Dr. Robert Dovenmuehle, who has been Director of Psychiatric Education of the WICHE in Boulder, Colorado, Dr. Robert Senescu, Chairman of the Department of Psychiatry at the University of New Mexico School of Medicine, and Dr. William Sheeley, Superintendent of the Arizona State Hospital and former Director of the General Practitioner Graduate Psychiatric Education Program of the American Psychiatric Association.

Dr. Sheeley will speak on "Emotional Problems of Teenagers" and "Community Resources Available to the Physician Managing Psychiatric Disorders"; Dr. Dovenmuehle on "Depression" and "Geriatric Psychiatry"; and Dr. Senescu on "Interviewing Techniques" and "Summary with Practical Applications."

The program on the 5th is open to all people interested in Alcoholism and Community Mental Health programs. Dr. William Sears, Psychiatric Consultant at the State Hospital, and Dr. John Abrums, Albuquerque, an Internist who is Director of the Turquoise Lodge of the State Commission on Alcoholism, will speak on Alcoholism.

There is no registration fee. The Town House Motel and the Palamino Motel in Las Vegas will serve as headquarters. Lectures and luncheons will be held at the State Hospital. For information about local arrangements or motel reservations write Dr. Carl Hallford, 720 University Ave., Las Vegas, New Mexico.

To Hold Medical Careers Conference

An Area Medical Careers Conference for junior and senior high school students, their parents and premedical club sponsors will be held in the Liberal Arts Building, Texas Western College, on Friday, March 4. Students from the entire Trans-Pecos area — including Brewster, Culberson, El Paso, Jeff Davis, Hudspeth, Loving, Pecos, Presidio, Reeves, Terrell and Ward counties — will be invited.

The program, which will include a tour of Thomason General Hospital, will consist of a panel discussion by representatives of the El Paso County Medical Society, the El Paso District Dental Society and leaders from other medically related groups on the recommended curriculum in high school and college, the medical college aptitude test, available loans and scholarships and medical careers. A film entitled "Design for Life" will be shown. The Conference will also include section meetings in medicine, ophthalmology, dentistry, medical technology, nursing, therapy, veterinary medicine and pharmacy. The Conference will be sponsored by the El Paso County Medical Society, the El Paso Public Schools and Texas Western College.

The N. M. - West Texas Urological Society will meet April 2-3, 1966, in Albuquerque, according to an announcement by Dr. H. J. Beck, Albuquerque, President.


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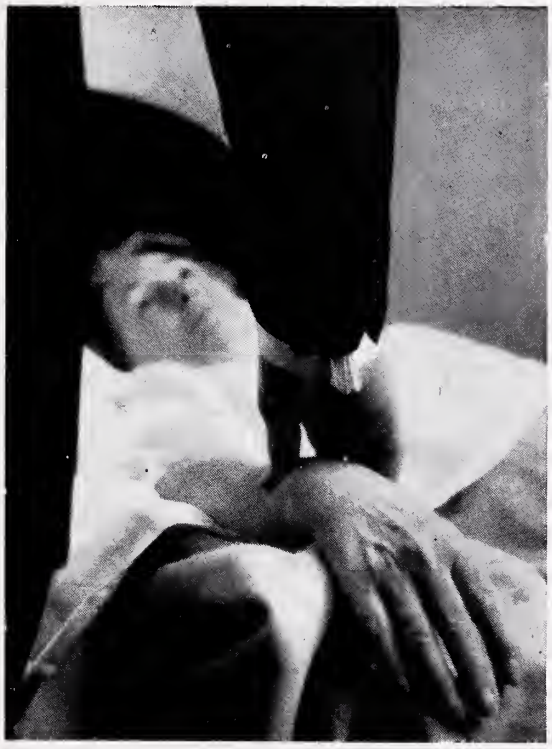

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Side effects: Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

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Southwestern MEDICINE

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and El Paso County Medical Society

IN THIS ISSUE

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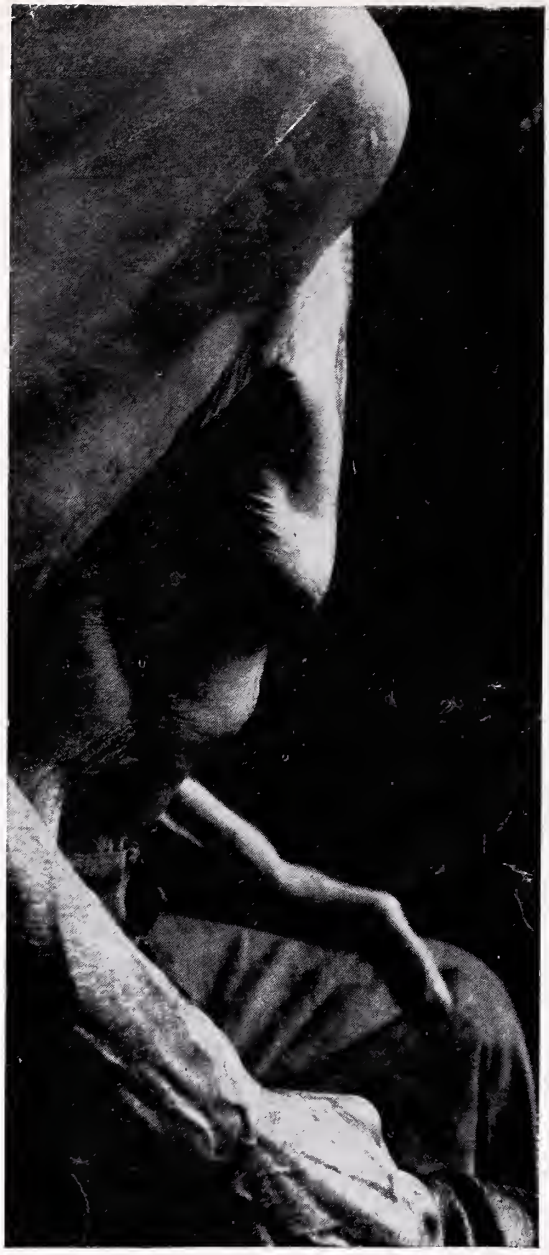
March, 1966

Founded 1916



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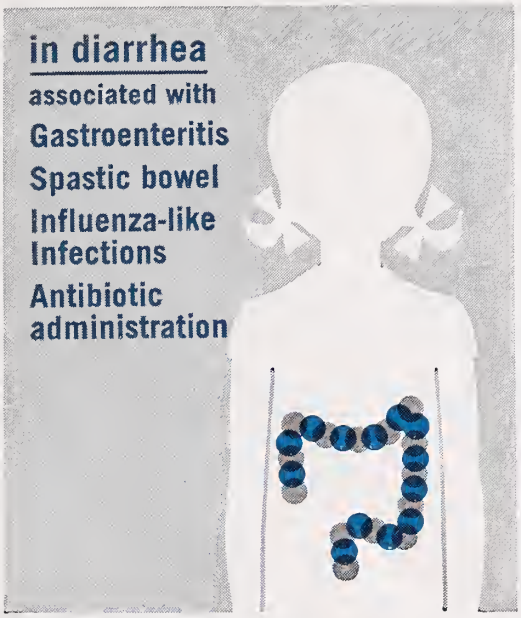

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1. Janssen, P. A. J., and Jageneau, A. H.: A New Series of Potent Analgesics: Dextro 2:2-Diphenyl-3-Methyl-4-Morpholinobutyrylpyrrolidine and Related Amides. Part 1: Chemical Structure and Pharmacological Activity, J. Pharm. Pharmacol. 9:381-400 (June) 1957.

2. Demeulenaere, L.: Action du R 1132 sur le transit gastro-intestinal, Acta Gastroent. Belg. 21:674-680 (Sept.-Oct.) 1958.

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Hypertension Revisited — 1966

Moderator and Editor: Lt. Col. Robert H. Moser, MC, Chief,
Department of Medicine, *El Paso*

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IV. Renal-Vascular Hypertension—Coarctation of the Aorta—Secondary Aldosteronism—Various Forms of Renal Hypertension—X-Rays—Laboratory Tests—Major Frank H. Chamberlin, MC

V. Treatment—Aims—Methods—Major Robert L. North, MC

I. Definition of Hypertension

(Major Ralph F. Wells, MC)

This discussion must be prefaced by acknowledging that the topic is expansive, the existing studies and opinions conflicting, and in general, the topic controversial.

A simple, practical scheme of classification of hypertension is by etiology, and this is basically the approach we shall use this evening. We may then see hypertension due to renal factors, hormonal or humoral factors, cardiac and vascular disorders, neurogenic factors, toxemia of pregnancy and finally, by exclusion, a disorder or perhaps a group of disorders characterized by the finding of diastolic hypertension and the absence of a primary etiologic process. This we shall specify as essential hypertension. Essential hypertension may be divided into two phases, benign and accelerated (malignant). In malignant hypertension the diastolic pressure may exceed 130 mm Hg; advanced retinopathy, encephalopathy or renal failure may be present.

"Mosaic Concept" of Page

Perera defines arterial hypertension as an elevation of the arterial blood pressure above the ranges encountered generally under routine or casual circumstances of recording. Herein lies the problem. The concept of essential hypertension is confused largely because of ignorance regarding the etiology and pathogenesis of the process. Page has proposed the "mosaic concept" in which a number of interrelated factors play a role, but the crux of the matter is at what level should a blood pressure be considered elevated and, if elevated, when is it clinically significant? The traditional division between normal blood pressure and hypertension is arbitrarily 150 mm Hg systolic and 100 mm Hg diastolic; the population falls in two groups on either side of this line of demarcation. By this standard a recent study of 15,000 persons revealed that one-third of the males over 40 and two-fifths of the females over 40 were hypertensive; the majority of men over 60 and women over 50 were hypertensive.

The Pickering View

Opposed to this view is the concept of Pickering who feels that essential hypertension represents the upper end of a distribution curve showing continuous variation with no evidence of two distinct populations. He feels that with advancing age the systolic and often the diastolic blood pressure increases, but that this phenomenon has no clinical or pathological significance. Compatible with Pickering's ideas are the results of studies by Masters and Jaffe who reviewed 74,000 World War II industrial workers, ages 16-64. The arbitrary lower limit of systolic hypertension was considered 140-190 mm Hg depending on age and sex; diastolic hypertension was set at 90-110 mm Hg again depending on age

and sex. Their observations made no allowance for qualitative changes in the vascular bed nor for genetic consideration.

Pickering's work has been severely criticized by Sir Robert Platt who felt the data was improperly analyzed and the genetic implication overlooked. He felt that Pickering's data showed neither a bimodal curve or age-related progression. Platt conducted a prospective study of siblings and parents of 75 patients with essential hypertension; he concluded that essential hypertension had a multifactorial genetic pattern with three phenotypic expressions reflected in a distribution curve. Clinical states consisted of normal, moderately hypertensive and severely hypertensive. In middle age, siblings of hypertensives show a rise in blood pressure clearly distinguishable from the slight rise in pressure that takes place with age in the normal population.

It is thus difficult to give a precise definition of hypertension or set precise numerical limits for its diagnosis. The point has been made that when there is evident organic change (retinopathy) or malfunction of a target organ system (renal dysfunction), generalized arteriolar disease is present. These arteriolar changes in general correlate with the degree of blood pressure elevation, and therefore, we are obliged to establish arbitrary limits to insure that treatment will be initiated before target organs are affected.

The diagnosis of essential hypertension is often complicated by the difficulty in establishing the "true" blood pressure of a given individual. A single blood pressure determination is not adequate; physical and psychic factors may introduce artifacts. This has led to the adoption of corrective factors, altered techniques of sphygmomanometry and the convention of recording serial (diurnal and nocturnal) blood pressure determinations by medical personnel other than the patient's own physician in an effort to identify the characteristics of the individual's blood pressure "profile." However, any random elevation of blood pressure is considered to be significant. The importance of recording the pressure in all four limbs of the hypertensive patient to exclude coarctation or other vascular anomalies is well established.

Perhaps of equal importance with documenting the blood pressure is the accurate and detailed appraisal of funduscopic findings. The Keith-Wagener classification remains the basis for grading hypertensive retinopathy. Brust has reviewed the normal anatomy of the fundus. After second branching, arteries of the fundus lose their muscular coat and may properly be called arterioles. They course through the unmyelinated fibers of the nerve layer (stratum opticum) of the retina which lies just behind the internal limiting membrane. The vessels run parallel to nerve fibers centrifugally from the disc; this explains the linear distribution of the hemorrhagic exudate.

Lockhart and his co-workers have modified the Keith-Wagener system and suggest the following scheme which encompasses both arteriolar constriction and sclerosis of the retinal vessels. (1) Grade I constriction consists of mild narrowing of the arterioles with an A/V ratio 1:2, (2) Grade II, a ratio of 1:3 or milder, generalized narrowing plus focal constriction, (3) Grade III, a ratio of 1:4 or intense generalized or focal constriction, (4) Grade IV, thread-like appearance with actual disappearance of many arteriolar segments. Sclerosis is graded by alteration of light reflex and A/V crossing defects. Grade I consists of increased light reflex with disappearance of venules at point of crossing; Grade II-copper wire changes with loss of venous column on each side of the arteriole; Grade III-silver wire changes with marked venous defects; Grade IV-white fibrous cords replacing arterioles. Under the Keith-Wagener scheme, Grade IV changes must include papilledema.

There is a general correlation of retinopathy with severity of diastolic blood pressure; values of 130 mm Hg or greater generally are present with Grade IV retinopathy. With adequate antihypertensive therapy even Grade IV changes are to some degree reversible.

Another important technique of physical examination is auscultation of the abdomen in search of a bruit or murmur in the patient suspected of having renal vascular hypertension. In a recent review, high-pitched murmurs were considered pathognomonic of renal artery stenosis. The murmur intensity was variable; in several instances it had a continuous to-and-fro character; in five patients it was located in the flank or CVA. Most commonly the murmur is heard anteriorly.

Cardiac findings range from normal to left ventricular hypertrophy and/or frank congestive failure. One must also mention the neurofibromata and cafe au luit spots sometimes seen

with pheochromocytoma. Seeking stigmata of Cushing's Disease, hyperaldosteronism and advanced renal disease are routine procedures.

Many have raised the question — "Is there a hypertensive personality type?" Although one must assess the general personality structure of the patient, I feel that observations in this sphere are conjectural. Perhaps the patient with hyperthyroidism, pheochromocytoma or CNS lesion may reveal helpful personality characteristics.

Laboratory Procedures

At this point it would seem appropriate to discuss some of the diagnostic procedures used in evaluation of the hypertensive. Routine studies should include Hematocrit, Hemoglobin, WBC and differential, urinalysis (with careful examination of the sediment), chest film, excretory urogram and ECG. Other laboratory studies include blood urea nitrogen, serum electrolytes, 24-hour urine for protein, and (if preceding studies or history are suggestive) urinary vanillyl mandelic acid (VMA) and/or catecholamines. Certain provocative tests may be useful if indicated. These include the cold pressor, phentolamine and histamine tests. Finally several special procedures are now available. These include the radio-isotope renogram; differential renal function tests (Na and volume) originally as proposed by Howard (modified by Stamey), aortography and renal biopsy.

I have not discussed incidence, mortality rates and therapeutic trends. I have reviewed briefly some of basic physical findings and laboratory studies involved in evaluating the hypertensive. The definition of (significant) hypertension remains obscure, but I hope these preliminary remarks will place the following discussions in proper context.

II. Hypertension and The Central Nervous System

(Major Darrell S. Buchanan, MC)

Over 40 years ago Fishberg observed and described a syndrome of seizures and focal neurologic signs complicating advanced hypertension. He called this condition hypertensive encephalopathy and ascribed the nervous system changes to arterial spasm and cerebral edema. Classically the sequence of events in this condition is: rising arterial pressure — progressively more severe headache — confusion — seizures — focal neurologic signs — coma and death.

Recent clinical-pathologic studies have indicated that the majority of cases diagnosed as hypertensive encephalopathy are found to have some other structural or physiologic abnormality which accounts for the encephalopathy. Some of these are intracranial hemorrhage, cerebral infarction, vascular insufficiency, uremia and electrolyte imbalance. These complications of hypertensive vascular disease are of course familiar to us all.

Assessment of the role played by the central nervous system in the etiology of hypertension has proved quite difficult. Early experimental work was concerned with demonstrating the ability of stimulation or destruction within the central nervous system to produce changes in systemic blood pressure. As early as 1864 Goltz demonstrated that stimulation in the lateral horn of the cord of a spinal animal would produce vasoconstriction. Pressor responses were also observed when nociceptive peripheral stimuli were applied to a spinal animal. It is now well-established that under normal physiologic conditions arterioles are partially regulated by vasoconstrictor fibers which are controlled by central nervous system mechanisms located in the medulla oblongata. The medullary centers are influenced by many factors. The more important of these are the baroceptor mechanism in the carotid sinus and aortic areas, and the distention receptors in the lungs. Also it has been well demonstrated that vasopressor responses can be produced by stimulation of various regions of the cerebral cortex, deep nuclear structures and the hypothalamus. Medullary centers also influence heart rate and cardiac contractile force to complement their vascular effect in control of blood pressure. Certainly, much of the work which has been done on standard "physiologic preparations" (anesthetized, thorocotomized animals) leaves much to be desired. It is quite likely that the normal integrated function of the nervous system of animals is significantly more complex and responsive than the largely peripheral or secondary mechanisms revealed by such studies in what are actually reasonably "unphysiologic" animal preparations.

Central factors are felt by many to have a significant part in the etiology of essential hypertension. It has been postulated that essential hypertension initially represents a conditioned response with interpsychic, interpersonal, and cultural determinents. This idea is an attractive one, but experimental proof of such a mechanism is extremely difficult. Soviet neurophysiologists

steeped in the tradition of Pavlov publish a great deal on this thesis.

Central Nervous System Hypertension

Clinical correlation of hypertension with a number of central nervous system disorders has been made on numerous occasions. Some of these include:

Brain tumors
Concussion
Hydrocephalus
Postpneumonoencephalogram
Lead encephalopathy
Subarachnoid hemorrhage
Porphyria
Meningitis
Diphtheria
Diencephalic Syndrome
Epilepsy
Polio
Guillian-Barre Syndrome
Riley-Day Syndrome
Basilar-vertebral insufficiency or thrombosis
Intracranial hemorrhage
Tabes dorsalis
Paraplegia
Post IXth nerve section
Polyneuritis
Encephalitis

The general mechanisms which may account for the hypertensive response in many of these conditions are:

Increased intracranial pressure
Anxiety
Response to pain
Medullary ischemia or inflammation
Interruption of Buffer Nerves
Anoxia or hypercarbia
Seizure

The hypertension which occurs in all of these clinical contexts is either paroxysmal or transient. Critical evaluation of reports of sustained hypertension due to central nervous system pathology or experimental lesions has usually revealed a more feasible explanation for the blood pressure elevation. An exception would be the experiment in which buffer nerves are destroyed, but this is actually a peripheral mechanism.

Basilar Artery Insufficiency

My own experience has been that the only neurological condition seen frequently in which secondary hypertension is a frequent feature is basilar-vertebral insufficiency or thrombosis. One such patient was a middle-aged Negro man with basilar thrombosis and marked blood pressure elevation. No past history of hypertension could be obtained. Phentolamine tests on two occasions were unequivocally positive. The patient died after about 36 hours; at postmortem no pheochromocytoma could be found.

Patients I have observed who survived longer than two or three days had a tendency for pressure to return to baseline levels.

I believe that hypertension as a manifestation of increased intracranial pressure has been greatly oversold. In most of the cases I have seen, the blood pressure starts to rise only as a near terminal event, and it is usually associated with marked bradycardia, respiratory dysrhythmia and other signs of medullary decompensation.

In conclusion, there is no unequivocal experimental data to support a hypothesis that chronic hypertension is due to primary organic changes in the central nervous system. Neurologic disorders in hypertensive patients are much more likely the result of, rather than the cause of, elevated blood pressure.

III. Humoral Hypertension

(Major Jerry M. Earll, MC)

There is increasing evidence to indicate that most forms of hypertension have "humoral" origin. Obviously, kidneys, adrenals, and nervous system all play roles in hypertension; the clinician's task is to assess relative importance of each organ in its contribution to the hypertension of his particular patient. The "mosaic theory" of hypertension is a useful approach. Well-known disorders of the adrenal gland that may be directly responsible for hypertension are Cushing's disease, primary aldosteronism, hypertensive variants of the adrenogenital syndrome, and pheochromocytomas. It is true that these pathologic entities constitute a very small percentage of all hypertensive causes (perhaps 1%), but they provide the clinician with the challenge of a correctable form of high blood pressure. The stimulus to research into the etiology and pathogenesis of all forms of hypertension has been inestimable.

Pheochromocytoma

Although Bright suggested a humoral mechanism for hypertension 135 years ago, the first clear evidence of endocrine participation in hypertension resulted from investigation of pheochromocytomas. The incidence of pheochromocytoma

is somewhere between one and four per thousand hypertensive patients. Although the point has been debated, it is readily apparent that neither clinician nor patient can afford the time and expense entailed in studying every hypertensive patient for pheochromocytoma. The clinician must be familiar with symptoms which suggest pheochromocytoma. The usual differential includes essential hypertension, hyperthyroidism, diabetes mellitus and pyschoneurosis. Many patients complain of excessive diaphoresis. This may be related to interference with heat loss mechanisms. Diaphoresis is an unusual finding in essential hypertension. Vasomotor phenomena such as coldness and numbness of hands and feet, blanching of fingers, skin pallor or mottling, and intermittent loss of peripheral pulses are common. Three-fourths of patients have elevations of body temperature by 1°F or more. A large majority have normal cold pressor tests (systolic rise less than 20 mm Hg and diastolic less than 15 mm Hg). Approximately 40 per cent of patients with essential hypertension are normal. Postural hypotension and tachycardia are other excellent signals that there is something unusual about a particular hypertensive patient.

Remember when the patient is in an upright position it is unusual for both systolic and diastolic pressures to fall below those seen when horizontal. Also it is rare for pulse rate in the standing patient to rise more than 20 beats per minute. Glucose intolerance and glycosuria occur, probably because epinephrine mobilizes carbohydrate from liver and muscles. The basal metabolic rate is often elevated and patients may appear "thyrotoxic." Beware of hypertensive patients who complain of "spells" or "attacks." Common symptoms during "attacks" are headache; palpitation; pain in the chest, abdomen, back or extremities; nausea; vomiting; sweating and exhaustion. Gastrointestinal symptoms are present in about one half of these patients; infarction and hemorrhagic lesions of the bowel have been reported. Weight loss is common (again suggesting the hypermetabolic state of thyrotoxicosis). There is an increased incidence of pheochromocytoma in patients with neurobifromatosis.

Pharmacologic Tests

Standard pharmacologic tests are familiar to all physicians and both the phentolamine blocking test and the histamine provocative test are useful. They must be performed with appropriate precautions; deaths have been reported from both procedures. Measurement of plasma and urinary catecholamines has the advantage of conservatism and may be the tests of choice in any high-risk patient. The biochemistry of catecholamines is well worked out. Tyrosine is hydroxylated to form 3-4 dihydroxyphenylalanine (DOPA), which is decarboxylated to 3-4 dihydroxyphenylethylamine (dopamine). This is hydroxylated in the beta position to yield norepinephrine. The term "catechol" refers to the dihydroxybenzene structure. The metabolism of these substances is accomplished by ortho-methylation and oxidative deamination. Of a given intravenous dose, about five per cent is excreted in the urine as catecholamine, 30-50 per cent as metanephrine or normetanephrine, and 30-50 percent vanillylmandelic acid (VMA). Although there was some controversy earlier, measurement of any of these substances is probably adequate. The "best test" depends upon which determination is most accurately accomplished in your laboratory.

Approximately 80 per cent of pheochromocytomas originate in the adrenal gland; 95 per cent are located in the abdomen. Less than five per cent are malignant, and as many as 10 per cent may be bilateral. Although both children and adults show definite predilection for the right side, many more children have bilateral, multiple, or malignant neoplasms. The tumors may be found wherever sympathetic ganglia or chromaffin tissue are known to exist. Venous catherization with determination of plasma catecholamines is helpful in locating some tumors. One of the newest tests involves administration of tyramine which causes release of catecholamines from tissues.

Surgical removal of a pheochromocytoma requires the teamwork of internist and surgeon. Preoperative expansion of contracted plasma compartments with an alpha receptor blocking agent (phentolamine) results in a much smoother operative course. Postoperative hypotension can be a severe problem in the absence of blocking agents; such drugs should be discontinued before surgery. Postoperative utilization of vasopressor agents may be required.

Adrenal Cortical Diseases

The adrenal cortical contribution to hypertension is well established (Cushing's Disease, adrenogenital syndrome variants and primary aldosteronism). Primary aldosteronism is ap-

proximately equal in incidence to pheochromocytoma. Cardinal features are hypertension and hypokalemia; symptoms include episodic weakness, periodic paralysis, tetany, polyuria (chiefly nocturnal), polydipsia, paresthesias and headache. Edema is rarely seen; if present it should suggest an additional disease process. Characteristic laboratory findings are hypokalemia, hypernatrema, alkalosis, moderate-to-marked increase in urinary free aldosterone, mild intermittent or persistent proteinuria, persistently neutral or alkaline urine. In addition one finds large urine volume (low specific gravity unresponsive to water restriction or administered vasopression), loss of renal conservation of potassium at low serum levels, abnormally small amounts of sodium in sweat and EKG changes compatible with hypokalemia.

Muscle weakness, nocturnal polyuria, headache, and polydipsia are the four most common symptoms. Although retinopathy occurs in approximately one half the patients, accelerated hypertension has been reported in only one case. The ratio of female-to-male patients is 2.7:1; the youngest patient reported was a 3½-year-old girl. Ninety-one per cent of the patients had a single adenoma. Over two-thirds of the tumors weighed less than six grams.

Over one half of the patients have shown reduced carbohydrate tolerance. In four-out-of-five patients the electrocardiogram suggests hypokalemia. Plasma volume frequently is elevated.

The diagnosis of primary hyperaldosteronism is made by demonstrating excessive adrenal secretion of aldosterone under physiologic conditions which normally do not provoke aldosterone production. Excessive secretion can be detected by direct determination via isotope dilution techniques, by measurement of urinary metabolites; or indirectly, by assessing physiologic effects. The first two methods are available only at special research laboratories; most clinicians make the diagnosis by studying physiologic effects. The differential diagnosis resides in the investigation of hypokalemia and hypertension. It is most important to measure 17-hydroxysteroids; if these are normal many diseases of adrenal and extra-adrenal origin associated with hypokalemia will be excluded.

With normal 17 OH steroids and evidence of hyperaldosteronism, the differential diagnosis is narrowed to primary or secondary aldosteronism. It must be kept in mind that mild cases which are completely asymptomatic have been recognized and cured surgically. Serum potassium may be in low normal range for weeks. In this type patient, 150-200 mEq of sodium per day is given for one week; during the last three days, daily determination of serum K and Na is made. Decreased serum potassium will provide the diagnosis. Remember that the low salt diet ingested by most hypertensive patients will obscure hypokalemia. A sodium load in the normal person will reduce aldosterone production, but will fail to effect an autonomous tumor. In the autonomous aldosterone state, the continuous abnormal exchange of sodium for potassium leads to hypokalemia. A common problem in differentiation occurs in the patient receiving diuretics who becomes hypokalemic. Some patients who are sensitive to diuretics also may have aldosteronism. Sometimes these drugs must be discontinued for several weeks while patients are placed on normal salt intakes to rule out diuretic effect as the primary cause. A lowered serum sodium and blood volume implicate the diuretic.

Potassium wasting renal diseases must be ruled out. Pseudoaldosteronism occurs in some patients eating large amounts of licorice. Spironolactone will antagonize the effects of glycyrrhizinic acid, but glycyrrhizinic acid does not measure as aldosterone in the urine. Vomiting, poor intake, or gastrointestinal losses of potassium should be apparent by history.

Congenital aldosteronism is not associated with tumors, but with bilaterally hyperplastic or normal adrenal glands. This is probably a form of secondary aldosteronism and may develop due to abnormal function of the juxtaglomerular apparatus. Of patients operated upon, 70 per cent were cured, 25 per cent improved and 5 per cent unimproved. Secondary aldosteronism must always be carefully differentiated; a small group of patients with primary aldosteronism and severe secondary renal damage present difficulties in diagnosis.

IV. Renal and Renal-Vascular Hypertension

(Major Frank H. Chamberlin, MC)

This portion of the discussion is directed at one form of hypertension that is "curable" by surgery, specifically that of renal origin. In 1934, Goldblatt demonstrated that a hypertension could be produced by progressive constriction of the renal artery in experimental animals. Goldblatt

was stimulated in his work by the observation that there was a significant incidence of renal artery stenosis in hypertensive patients who came to autopsy. This was the original work in an area that has expanded to include the present-day search for answers to the questions: What is the lesion that produces hypertension? How is it generated? What is the mechanism of action? How can the clinician detect the renal origin of hypertension? These are some of the topics which I will cover in this section.

I will not cover the problem of parenchymal disease of the kidney such as chronic pyelonephritis, glomerulonephritis, the renal expression of systemic collagenoses, infiltrative diseases of the kidney (amyloidosis) and others. This discussion will center on renal lesions causing hypertension that can be corrected. Stamey discussed chronic pyelonephritis only to dismiss it saying, "The relationship between pyelonephritis and hypertension continues to be elusive and probably of little importance." This is a curious statement. In Palo Alto, Stamey had not seen a patient whose hypertension was caused by unilateral pyelonephritis (despite a 12-year experience during which 30 per cent of patients with bacteriuria of bladder origin had unilateral pyelonephritis). He also stated that 98 per cent of hypertensive patients referred for evaluation had sterile urine at cystoscopy. He felt that the relationship of pyelonephritis to hypertension was more a matter of degree of impaired renal function (azotemia) than a specific response to renal infection. Thus it would seem rather important to attack acute pyelonephritis with consummate vigor to forestall chronic disease with azotemia.

There is no clear figure on the incidence of renal-vascular hypertension. However it is estimated that approximately five per cent of unselected hypertensive patients will have occlusive change in one or both renal arteries. The two major categories of occlusive lesions are fibromuscular hyperplasia and atheromatous disease. Congenital abnormalities of renal vascularity other than fibromuscular hyperplasia are rare. Hypertension related to fibromuscular hyperplasia usually is found in young patients. Aortography reveals a characteristic irregular, beady appearance of the renal artery.

The bulk of lesions fall into the atheromatous category. In both groups, atheromata may be present bilaterally; the cardinal problem is which lesion (or both) is producing the hypertension? Or is either at fault? Unfortunately similar renal artery lesions can be found in normotensive subjects.

A significant research effort has been directed at investigation of the pathogenesis of renal-vascular hypertension. Attention has been focused on renal juxtaglomerular cells located around the afferent arteriole of the glomerulus. Granules within these cells have been shown by various biochemical and immunofluorescent techniques to consist of renin or a very similar substance. Renin is a proteolytic enzyme secreted by the kidney. A protein substrate synthetized by the liver is the substrate for the enzymatic action of renin. As a result of this proteolysis a decapeptide called angiotensin I is split off. This amino acid complex has little pharmacologic activity. Another enzyme called "converting enzyme" by Skeggs (identified as angiotensin activator by Page and his co-workers 20 years ago) splits histadyl leucine off the decapeptide; the resulting octapeptide, angiotensin II, is the active pressor agent. Some authors believe that this is the agent responsible for hypertension related to renal-vascular disease. The subsequent action of angiotensin II on the adrenal cortex provokes secretion of aldosterone. The properties of aldosterone are well known. The overall effect preserves intravascular fluid volume by retention of sodium; excretion of potassium and hydrogen occurs in compensation. Many authors believe most renal-vascular hypertension is related to "secondary aldosteronism."

The stimulus for release of renin is debatable. In very elaborate and precise experiments on the dog, Page and co-workers found that renin secretion is controlled by a renal baroreceptor rather than by ischemia. The experiment involved placing a band around the aorta above the renal arteries. When the mean perfusion pressure was reduced by as little as 5 mm Hg, increased renin secretion began within 60 seconds. The converse was also true; increasing mean perfusion pressure resulted in reductions of renin release. A small amount of renin was secreted continuously under the conditions of the experiments. This suggested to the investigators that a renal baroreceptor mechanism regulates renin secretion under normal circumstances, and arterial pressure tends to stabilize at a level at which renin secretion is minimal.

It has been known since 1938 that any large vessel partially constricted for a short distance must be occluded by at least 70 per cent in cross-sectional area before blood flow is altered distal to the obstruction. This has caused much difficulty in the evaluation of renal-vascular hypertension because there are many patients with hypertension and radiographically identifiable stenosis of the renal artery who do not have renal-vascular hypertension. Failure to consider this fact contributes to the high failure rate of surgery in "curing" hypertension when renal-vascular occlusion demonstrated by aortogram was the sole criterion for surgery. It is evident that precise tests are necessary to determine which patients have true renal-vascular hypertension.

"Routine" baseline studies have been discussed. The rapid-sequence excretory urogram has replaced more conventional techniques in evaluating hypertensive patients. The radiopaque substance is injected with a large needle in 30 seconds, and then films are taken at 1, 2, 3, 4, 5, 10, and 15 minutes. Parameters used to evaluate renal lesions are: (1) decrease in longitudinal diameter of one kidney by 1.5 cm or greater and (2) delay in appearance time of one minute or more. By these criteria, in one report, 39/42 patients with renal-vascular hypertension had abnormal intravenous pyelograms.

The next group of tests is reserved for patients particularly suspected of having renal-vascular hypertension. Clinical clues are: malignant hypertension of abrupt onset in a previously normotensive person; hypertension in patients younger than 35 years of age; sudden acceleration of vascular disease; abrupt development of hypertension after the age of 35; symptoms of atherosclerosis preceding the onset of hypertension and epigastric bruits.

Split Function Tests

Because the involved kidney has demonstrated increased avidity for water, many tests have been devised for measuring water absorption by the tubules of the involved kidney in the form of "split renal function" tests. The first of these was the Howard Test; this called for measurement of sodium and water on each side. Stamey modified the test and measured differential concentrations of poorly reabsorbed substances such as para-aminohippuric acid (PAH), inulin, creatinine and urea. Rapoport further modified the procedure by comparing ratios of urinary sodium and creatinine. The Stamey and the Rapoport tests are used most often. The Howard Test is not as reliable because the substances measured are both reabsorbed. The Rapoport Test has the advantages of measuring a ratio of normally excreted substances and the artifact of ureteral obstruction is eliminated; volumes are not used in the calculation. When the capability for accurate split renal function tests is present, they should be done on all renal lesion suspects.

Radioisotope Renogram

The radioisotope renogram is a very effective method for evaluating renal function. There are three components to the renogram. (1) The initial or vascular spike completed within 20 seconds. (2) A secondary rise a few seconds later is rapid and has an amplitude one third that of the vascular spike; it lasts for 4-6 minutes and records the functional capacity of the kidney. It is produced by a combination of vascular inflow, tubular secretion and evacuation of urine after a couple of minutes. (3) The excretory phase is represented by a sharp exponential drop in the curve which is completed within 5-10 minutes. The radioisotope renogram combined with the rapid-sequence intravenous pyelogram constitute reliable screening procedures to define a unilateral renal lesion.

The next step in evaluating suspected renal lesions is aortography with the bolus of dye introduced above the renal arteries followed by rapid exposure examinations to observe the filling of renal vessels. This can be done by two methods, translumbar or retrograde femoral. Both techniques are potentially dangerous; aortograms should be done only when split function and/or renogram studies are abnormal. Also, a strong clinical suspicion is adequate reason to pursue a renal lesion by aortography.

Renal Biopsy

Until recently, renal biopsy has not enjoyed much favor in the evaluation of hypertension of renal origin because its main value lies in the diagnosis of parenchymal lesions. There is no histologic picture typical for renal-vascular hypertension. However, Page has reported that in 52 consecutive patients on whom bilateral biopsies were done, histologic abnormalities were present bilaterally in all. Thus, renal biopsy can provide information on histologic appearance of smaller renal arteries that may escape detection by aorto-

graphy. Abnormality of the smaller renal arteries may be related to hypertension, which persists after surgical correction of main renal artery occlusive disease.

Recent tests which still have not found their proper place in the scheme of things are: (1) Measurement of plasma renin levels (reported by Conn) can be useful in identifying secondary aldosteronism. The theory is simple—if the kidney is secreting excess renin which in turn is producing aldosterone release, this can be measured. (2) McPhaul and associates at Wilford Hall USAF Hospital have reported finding a pressor agent, as yet unidentified, in renal vein blood of hypertensive patients in whom surgical correction of renal vascular lesions resulted in "cure" of hypertension. The technique requires differential blood specimens from the renal veins. It is not a simple procedure. (3) The final test is the angiotensin-infusion test (described by Kaplan). This also depends on the renin-angiotensin system. If a renal lesion exists, increased renin is present and increased amounts of angiotensin II will be produced. Vasopressor receptor sites will be less responsive to the exogenous pressor agent, angiotensin. The test is simple. It involves calculating the amount of angiotensin required to raise diastolic pressure 20 mm Hg. A screening test is advised in which a known amount of angiotensin is injected and the effect monitored. A positive test consists of either: (1) large amounts of angiotensin being necessary to elevate the diastolic pressure 20 mm or, (2) little effect from a test dose.

Surgical Treatment

Treatment in the eyes of the internist is simple. There are two approaches; reconstructive vascular surgery can be attempted or the offending kidney can be removed. The internist is obliged to present positive evidence that the renal vascular lesion is the cause of the hypertension. Simple demonstration of occlusive disease may not be sufficient.

From the aspect of the surgeon there are several approaches. He may remove the kidney. He may elect to resect the occluded part of a vessel and anastomose; use a patch graft or prosthesis. There have been reports of bilateral reconstructive vascular surgery. Long-term results are often disappointing. The surgical approach to renovascular hypertension is not a closed chapter.

In summary, renal-vascular disease is a rare cause of hypertension, but it may be one of the few curable situations; it is worthwhile to investigate and discover. Problems in making the diagnosis revolve around establishing that indeed the kidney is the cause of the hypertension. Then one must determine which kidney is the offending member, or if both are involved. There is no set formula. All patients with severe hypertension should have a rapid sequence excretory urograph, and if available, radioisotope renogram. Patients who are considered good candidates for renovascular hypertension from a clinical (historical) aspect should then have split renal functions and aortography. The newer tests may be helpful, but are still in formative stages; they are not available to the majority of clinicians, and their usefulness has not been clearly established.

V. Therapy of Hypertension

(Major Robert L. North, MC)

The finding, on physical examination, of high blood pressure is but another fact in a world full of facts. Previous participants in this symposium have indicated some of the difficulties in defining hypertension and hypertensive disease, the problems in establishing causes and effects, and the uncertainty in prognosis for individual patients who present this physical finding. It is no part of my discussion to quibble about a millimeter or two of mercury, but I do wish to emphasize strongly that even relatively minor degrees of persistent blood pressure elevation ultimately cause an increase in morbidity and mortality, regardless of etiology. This fact leads to a very simple, fundamental principle of therapy, and one which applies to every case; blood pressure should be reduced to levels as near normal as possible for as much of every day as possible. This simply-stated, but too infrequently achieved, therapeutic aim cannot be overemphasized.

The means by which this aim may be accomplished are varied and imperfect; there is no ideal therapeutic agent or technique. Surgical procedures, e.g., in patients with coarctation of the aorta, pheochromocytoma or carefully selected patients with major renal-vascular obstructive lesions may be highly effective, but will not be discussed here because of time limitation. We are concerned in this discussion with drug therapy.

Hypertensive patients must be evaluated thoroughly from the etiologic standpoint and from the aspect of deterioration in end-organ function (especially brain, heart, and kidneys) to guide

therapy and gauge prognosis. Many elaborate schemes for grading the severity of hypertensive disease have been devised; nevertheless, the serial casual measurement of the systemic blood pressure by an ordinary sphygmomanometer is the most important single criterion for therapy.

Psychotherapy, establishment of a program of adequate exercise and rest, and maintenance of ideal body weight are measures of more or less importance in every hypertensive patient and will occasionally produce surprising results when used alone. Almost every patient with hypertension will benefit from sodium restriction in the diet. We generally aim at a diet containing 20 mEq sodium and consider ourselves fortunate if the patient's average intake is 40 mEq or less. These measures are important and highly effective, but most patients will require the addition of one or more drugs to his therapeutic program.

Hypertensive disease is a dynamic process, and as we have seen, probably each case is multifactional in cause. The therapeutic program must also be dynamic, continually subject to revision, and tailored to the needs of the individual patient. Patients with significant, sustained diastolic hypertension are usually asymptomatic in the earlier stages of the disease: it may take 10-20 years to bring about the inevitable deterioration in cerebral, cardiac or renal function whereby the hypertensive patient ultimately sickens and dies. We are firmly convinced that the effective lowering of blood pressure by drugs markedly improves the outlook for these patients, but this is often accomplished only at the expense of drug-induced morbidity. Excellent rapport with the patient is essential, since he must understand and accept the need for long-continued and sometimes troublesome treatment.

The following table is a general guide for the treatment of ambulatory, middle-aged, essential hypertensive patients. Patients with severe degrees of hypertension or with acute complications should be hospitalized for treatment.

The premium in drug therapy is placed not on mere hypotensive effect, but upon predictability of action, freedom from side effects and absence of drug tolerance and tachyphylaxis. Presently available drugs are highly effective, but none is ideal.

The benzothiadiazine (thiazide) group of diuretics are all derivatives of a basic sulfonamide nucleus and differ mostly in potency per unit weight rather than in any essential pharmacologic effect. For most patients, chlorothiazide is well tolerated, effective and least expensive. The exact means by which these drugs lower blood pressure is not known. Any effective diuretic will usually produce a temporary drop in blood pressure in hypertensive patients, but the sustained effects of the thiazide drugs are not directly correlated with weight loss, blood volume decreases, or change in cardiac output. Probably an altered vascular reactivity to a variety of stimuli is the basic mechanism. There are extremely valuable adjunctive agents for potentiating the effects of other antihypertensive drugs even when not en-

Table I

DIASTOLIC BLOOD PRESSURE	INITIAL THERAPY	ADJUNCTIVE THERAPY
90 to 110 mm Hg	Thiazide diuretic	Rauwolfia
110 to 130 mm Hg	Thiazide diuretic	L — methyldopa
	+ guanethidine	pargyline, hydralazine
	HOSPITALIZATION	
130 mm Hg	Bed rest, thiazide diuretic, guanethidine	As above
Hypertensive emergencies	Rest, sedation, parenteral reserpine	Sodium nitroprusside or intravenous ganglionic blocking agents (trimethaphan camphor sulfonate, pentolinium or hexamethonium)

tirely effective alone. They may produce severe hypokalemia, hypochloremia and alkalosis, hyperuricemia and gout; they may aggravate diabetes or pre-diabetes, and a variety of less common, but serious, adverse effects have been reported.

One must be alert to the propensity for producing clinically important hypokalemia which these drugs display. Among other serious effects, hypokalemia may be directly responsible for the precipitation of digitalis intoxication, and there is some evidence that the impairment in glucose metabolism in diabetes and pre-diabetes is worsened by hypokalemia. It has been our practice in the past to almost routinely prescribe supplementary potassium chloride in the form of tablets or elixir, 1-1.5 gms per 500 mg of chlorothiazide or equivalent. Small bowel ulcerations may be produced by the high local concentration of KCl released from tablets and absorption is unpredictable. The elixir is a less convenient formulation and may cause nausea but is preferred nonetheless. Recently combined treatment with triamterene in selected patients has lessened the risk of hypokalemia without use of supplemental dietary potassium. The addition of another drug to the therapeutic program of course entails another set of potential ill effects. At present then, the wisdom and feasibility of attempting to prevent thiazide-induced hypokalemia by the foregoing means are open to question, but once clinically-manifest hypokalemia appears, therapy must be prompt and vigorous. We rarely give more than 500 mg chlorothiazide twice daily or its equivalent.

Reserpine is the principal active ingredient in rauwolfia and the purified drug is the preparation most often prescribed. This agent depletes tissues of norepinephrine, reducing total body stores of this neurotransmitter. It also has marked sedative and tranquillizing effects which are useful in an occasional patient. Severe and sometimes suicidal depression may occur; bradycardia and cardiac arrhythmias may develop, and weight gain is often a problem. Maximum oral dose for long-term therapy is 0.5-0.75 mg daily. I do not like to use reserpine with guanethidine because of the potential for serious arrhythmias. Intramuscular reserpine in doses of 1-10 mg at intervals of 1-4 hours is a very effective agent for moderately rapid lowering of blood pressure in malignant hypertension or hypertensive emergencies. In this dose range and by this route, it may aggravate peptic ulcer disease, and occasionally causes severe hypotensive reactions.

The ganglionic blockers and guanethidine produce a blockade of sympathetic neural homeostatic reflex activity. Guanethidine depletes tissue norepinephrine, and produces a peripheral blockade at the effector site. These two effects are not necessarily directly related. The older ganglionic blockers are rarely used nowadays because of the severe side effects due largely to parasympathetic blockade. In the resting, supine or hypervolemic patient when there is little sympathetic vasoconstrictor tone, the agents are less effective, although variable reduction in blood pressure due to depressed cardiac output may be seen. When the patient is erect, or physically active, the hypotensive effect may be extreme. Guanethidine is extremely potent and relatively well accepted by patients. We usually begin outpatients on 10-12.5 mg daily and increase the dose by similar increments at weekly intervals until the desired effect is obtained. Overdosage may take several days to resolve and postural hypotension is common. Guanethidine causes fluid retention and may precipitate congestive failure in susceptible individuals. Diarrhea, failure of ejaculation and a peculiar type of myasthenia are among the other side effects.

Time does not permit discussion of the monamine oxidase inhibitors such as pargyline or the decarboxylase inhibitor, alphamethyldopa, which are occasionally useful moderate antihypertensive agents. Their mechanism of action in lowering blood pressure is unknown and may not be related to the relatively weak in vivo enzymatic blockade they produce. Similarly, hydralazine is only occasionally useful, causes many side effects, some serious, and will not be discussed in detail.

The treatment of severe, or complicated, hypertension and hypertensive emergencies is a topic in itself and will not be further discussed here.

The subject of hypertension will continue to intrigue and fascinate clinician and investigator. We have learned a great deal; we have a great deal yet to learn.

Dr. C. A. Robinson Elected President of TMA District I

Dr. C. A. Robinson, Kermit, was elected President of District I of the TMA at a one-day joint meeting of District I and the El Paso County Medical Society in El Paso, February 5, 1966.

Other new officers are Dr. George W. Iwen, El Paso, President-Elect, and Dr. Ira A. Budwig, El Paso, Vice-President. Dr. Budwig is the retiring President. Dr. Mario Palafox, El Paso, was re-elected Secretary-Treasurer. Dr. Russell Holt, El Paso, continues as Councilor.

Dr. David Wade, Austin, President of the TMA, was the banquet speaker. Mrs. Russell L. Deter, El Paso, President of the TMA Woman's Auxiliary, was an honored guest at the banquet.

The 1967 meeting will be held February 4 in Pecos. Dr. James D. Murphy of Ft. Worth, President-Elect of the TMA, will be the principal speaker.

Scientific speakers at the El Paso meeting were: Dr. Louis W. Breck, Dr. Victor M. Blanco, Dr. K. Zolfoghary, and Dr. Jose Roman, Jr., all of El Paso.

Mrs. Roy F. Kemper, Coleman, Texas, Regional Vice-President of the TMA Auxiliary, spoke at a luncheon for wives. Mrs. Jesson L. Stowe, El Paso, was in charge of the Auxiliary program.

Dr. Robinson

Dr. Robinson was born in Golden, Texas, received his pre-medical education at the University of Utah and was graduated from Baylor University College of Medicine. He interned and did his surgical residency at Parkland Hospital from 1933 to 1935. He began the practice of medicine in Gladewater, Texas, in 1935. He moved to Kermit in 1937.

He received his orthopaedic specialty training from 1950 to 1953 on the Northwestern program and has been a member of the American Academy of Orthopaedic Surgeons since 1957. He is also a Fellow in the American College of Surgeons and in the International College of Surgeons.

His wife is Dr. L. Rose Robinson, who was a classmate at Baylor. The Robinson's were married in Gladewater in 1935 and have practiced together since then. Dr. Robinson's wife has limited her practice to Pediatrics since 1940. At present, she is Chief-of-Staff of the Winkler Memorial Hospital.

The Robinsons are not the only husband-wife medical team in Kermit. Dr. Harper Peddicord and his wife, Dr. Orene Peddicord, are in General Practice there. Dr. Harper Peddicord is President of the Reeves-Ward-Winkler-Loving-Culberson-Hutchinson Counties Medical Society. Dr. Orene Peddicord is Secretary of the local Medical Society.

Urologists to Meet in Albuquerque, April 2-3

Dr. Paul Peters, Dallas, Assistant Professor of Urology at the University of Texas Southwestern Medical School, and Dr. Wadi Suki, Instructor in the Department of Internal Medicine at Southwestern, will speak at the annual meeting of the New Mexico—West Texas Urological Society in Albuquerque, April 2-3, 1966, in the Bernalillo County Indian Hospital. Dr. H. J. Beck, Albuquerque, is President.

The meeting will run from 9 a.m. through 4:30 p.m., with cocktails and dinner at 7 p.m., April 2, and from 9 a.m. to 12:30 p.m. April 3.

Dr. Peters will speak on "The Treatment of Renal Failure, Including Renal Transplantation" and "Detection of Genito-Urinary Anomalies During Intra-Uterine Life and in the Newborn Nursery". Dr. Suki will speak on "The Consequences of Urinary Tract Obstruction" and "The Mode of Action of Diuretics". There will be a round table luncheon on the 2nd at noon. From 9 to 11 a.m., April 3, there will be a Pyelogram Hour with interesting cases.

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Contraindications: Previous allergic or idiosyncratic reactions to meprobamate or meprobamate-containing drugs.

Precautions: Careful supervision of dose and amounts prescribed is advised. Consider possibility of dependence, particularly in patients with history of drug or alcohol addiction; withdraw gradually after use for weeks or months at excessive dosage. Abrupt withdrawal may precipitate recurrence of pre-existing symptoms, or withdrawal reactions including, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, the dose should be reduced and operation of motor vehicles or machinery or other activity requiring alertness should be avoided if these symptoms are present. Effects of excessive alcohol may possibly be increased by meprobamate. Grand mal seizures may be precipitated in persons suffering from both grand and petit mal. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side effects: Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

Usual adult dosage: One or two 400 mg. tablets three times daily. Doses above 2400 mg. daily are not recommended.

Supplied: In two strengths: 400 mg. scored tablets and 200 mg. coated tablets.

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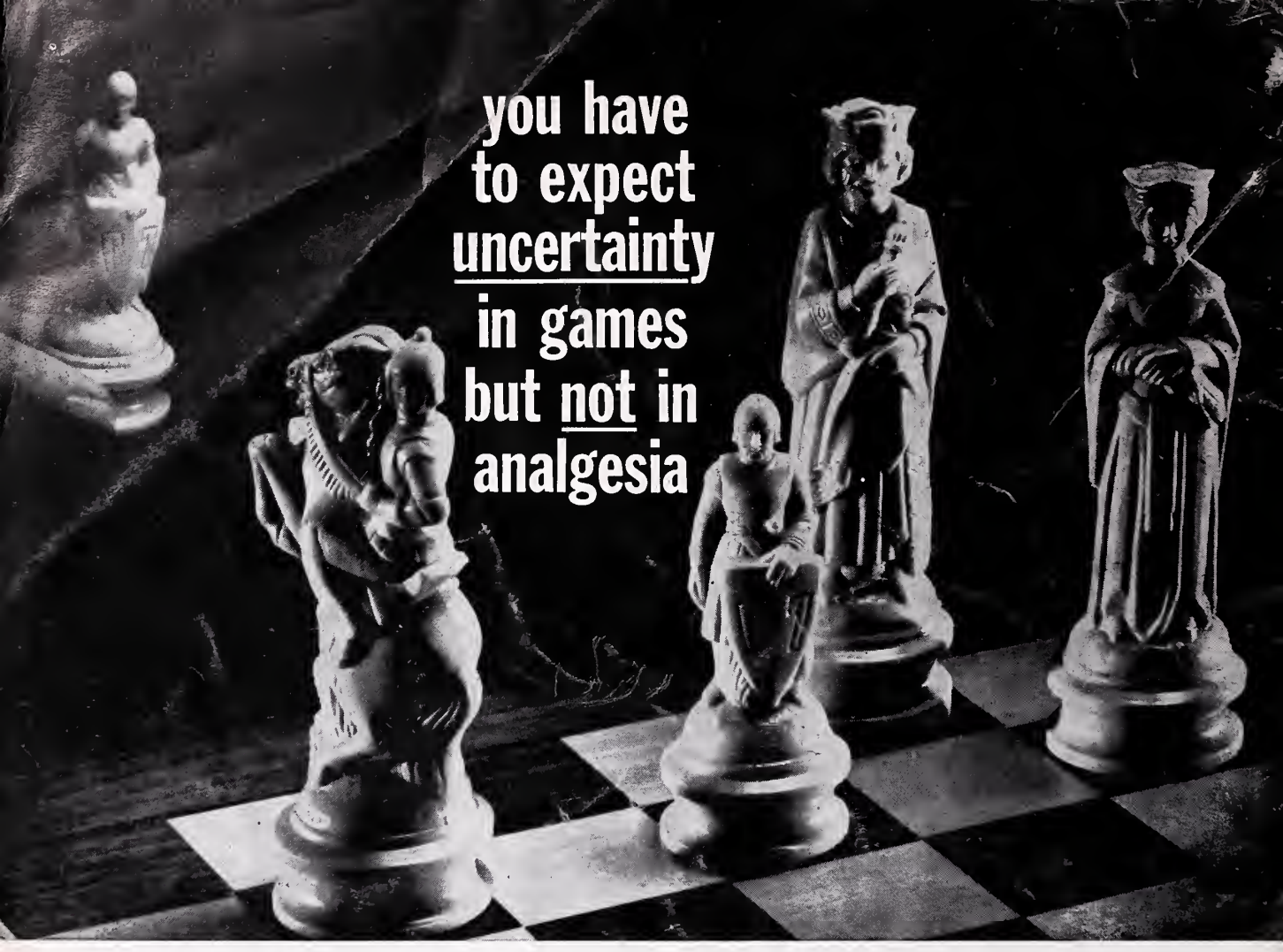

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Southwestern MEDICINE

Official Journal of the Southwestern Medical Association,
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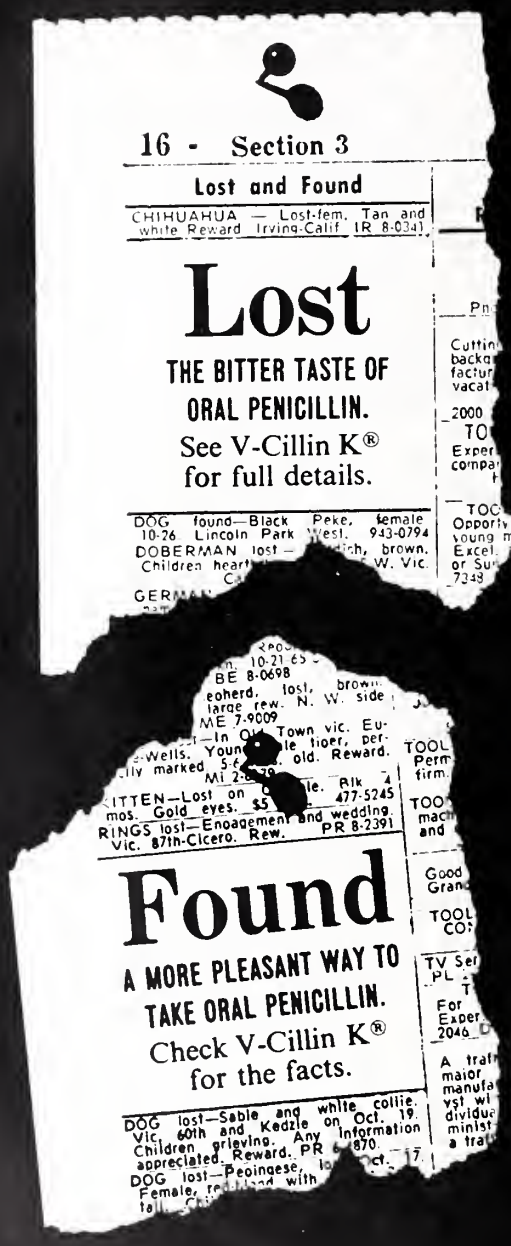
and El Paso County Medical Society

IN THIS ISSUE

VOL. 47, NO. 4

April, 1966

Founded 1916



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The U. S.-Mexico Regional Medical Journal Serving West Texas, New Mexico, Arizona, Nevada and Northern Mexico

Official Journal of

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VOL. 47 APRIL NO. 4

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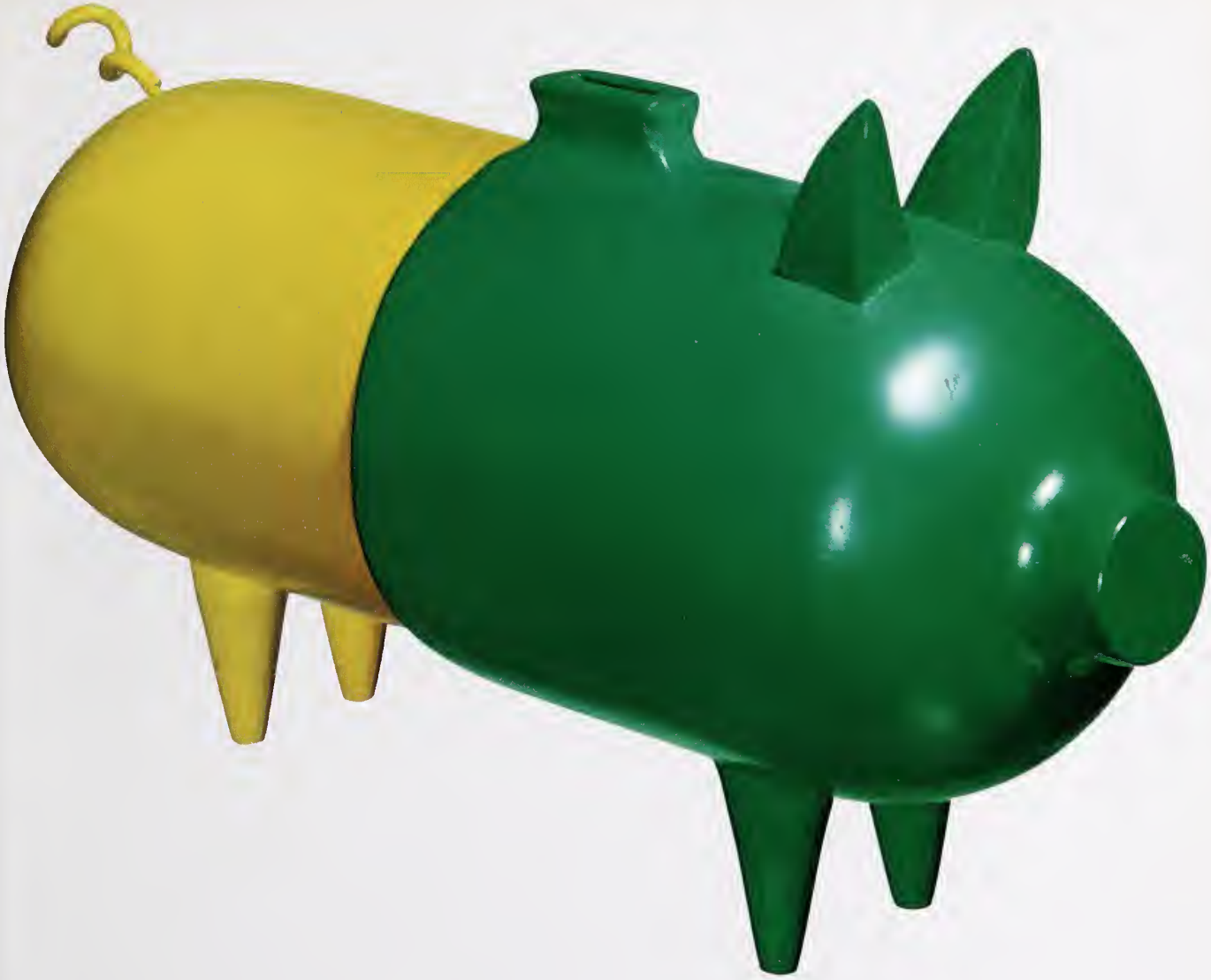

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BRIEF SUMMARY: Blurring of vision, dry mouth, difficult urination, and flushing or dryness of the skin may occur on higher dosage levels, rarely on usual dosage. Administer with caution to patients with incipient glaucoma, or urinary bladder neck obstruction. Contraindicated in acute glaucoma, advanced renal or hepatic disease, or a hypersensitivity to any of the ingredients.

REFERENCES: 1. Vollmer, H.: Arch. Neurol. and Psychiat., 43:1057, 1940. 2. Morrissey, J.H.: J. Urology, 57:635, 1947. 3. Krantz, J.C., Jr., and Carr, C.J.: Pharmacological Principles of Medical Practice, 2nd ed., Baltimore (1954), 552.

*This one at Westover, elegant Colonial Virginia plantation, located on the James River near Richmond. Built in the early 1730's by William Byrd II, founder of Richmond, it is now the home of Mrs. Bruce Crane Fisher.

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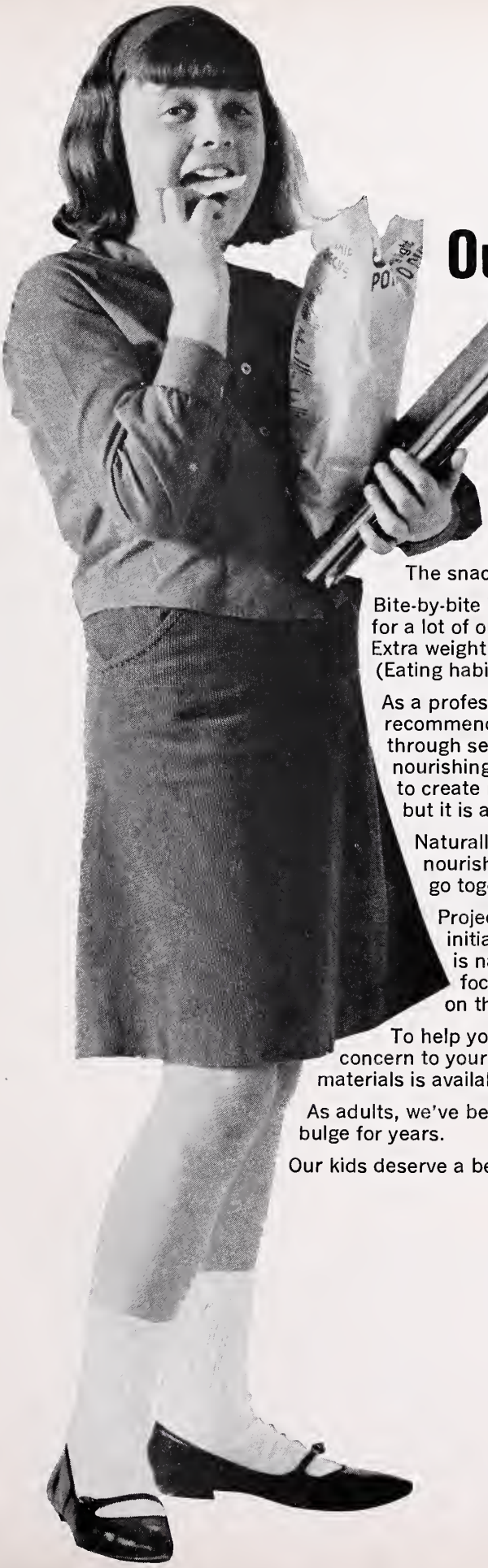

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Usual adult dosage: One or two 400 mg. tablets three times daily. Doses above 2400 mg. daily are not recommended.

Supplied: In two strengths: 400 mg. scored tablets and 200 mg. coated tablets.

Before prescribing, consult package circular.

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N. M. Medical Society To Hold 84th Annual Meet In Albuquerque

The 84th Annual Meeting of the New Mexico Medical Society will be held in Albuquerque, May 10-13, 1966, with Business Sessions in the Western Skies Hotel and Clinical Sessions in the Student Union Building of the University of New Mexico.

Speakers on the clinical program will be Dr. Philip P. Ellis, Associate Professor and Head of Ophthalmology at the University of Colorado Medical Center in Denver; Dr. Theodore N. Finley, Associate Professor of Medicine at the University of New Mexico School of Medicine; Dr. Alexander L. Kisch, Assistant Professor of Medicine at the University of New Mexico School of Medicine; Dr. Jay Philip Sanford, Professor of Internal Medicine at the University of Texas Southwestern Medical School at Dallas; Lt. Col. Walter E. Switzer, MC, Chief of the Clinical Division of the U. S. Army Surgical Research Unit at Brooke Army Medical Center at Fort Sam Houston, Texas; Dr. William H. Tooley, Associate Professor of Pediatrics and Staff at the University of California Medical Center at San Franciso; Dr. Max H. Weil, Associate Professor of Medicine and Director of the Shock Research Unit at the University of Southern California School of Medicine at Los Angeles; Dr. Leo E. Hollister, Associate Chief of Staff at the Veterans' Administration Hospital at Palo Alto, California; and Dr. Charles C. Bollinger, Chief of Obstetrics and Gynecology for the U. S. Public Health Service at the Indian Hospital in Gallup, New Mexico.

The House of Delegates will meet May 10 and 11, and an Orientation Course for new members will be held May 11. The Clinical Sessions are scheduled for May 12 and 13.

Officers of the Society are Dr. Robert P. Beaudette, Raton, President; Dr. Thomas L. Carr, Albuquerque, President-Elect; Dr. Emmit M. Jennings, Roswell, Vice-President; and Dr. John D. Abrums, Albuquerque, Secretary-Treasurer. Members of the Program Committee are Dr. Solomon Papper, Albuquerque, Chairman; Dr. Andrew M. Babey, Las Cruces; Dr. Harry D. Ellis, Santa Fe; Dr. Robert S. Stone, Albuquerque; Dr. Earl B. Flanagan, Carlsbad; and Dr. Reginald H. Fitz, Albuquerque.

The complete program is as follows:

Business Meetings

Western Skies Hotel

Monday, May 9

2:00 p.m. Council Meeting

Tuesday, May 10

1:00 p.m. Registration

2:00 p.m. House of Delegates, First Session
Reference Committee Meetings

Wednesday, May 11

Orientation Course for New Members
Presiding: Emmit Jennings, M.D., Roswell, Chairman, Orientation Course

8:00 a.m. Registration for Orientation Course

8:45 a.m. Welcome
Robert P. Beaudette, M.D., Raton, President, New Mexico Medical Society

9:00 a.m. "Narcotics"
William F. Quinn, M.D., Los Angeles, Chairman, Narcotics Commission of California

9:30 a.m. "Medical Negligence"
Albert G. Simms, M.D., Albuquerque,

Member, Medical-Legal Committee, New Mexico Medical Society

9:50 a.m. "The Doctors In Court"
Robert Taichert, LL.B., Albuquerque, Legal Counsel, Bernalillo County Medical Association

10:15 a.m. Coffee Break

10:30 a.m. "Critical Dimensions"
AMA Film

11:00 a.m. "Resources of the Medical School to the Practicing Physician"
Reginald Fitz, M.D., Albuquerque, Dean, University of New Mexico School of Medicine

11:20 a.m. "Sanity Laws and Methods of Commitment"
Hon. Robert W. Reidy, Albuquerque, District Judge, Bernalillo County

11:45 a.m. "Medical Ethics"
R. C. Derbyshire, M.D., Santa Fe, Secretary-Treasurer, New Mexico Board of Medical Examiners

12:05 p.m. Questions and Answers

12:30 p.m. Luncheon
(Courtesy of New Mexico Medical Society for New Members and Guest Speakers)
Speaker: William F. Quinn, M.D.

2:30 p.m. House of Delegates, Second Session

6:00 - 7:15 p.m. Convention Cocktail Party
(Courtesy New Mexico Medical Society)

7:30 p.m. New Mexico Political Action Committee Banquet
Western Skies Hotel

General and Clinical Meetings
Student Union Building
University of New Mexico
Thursday, May 12

GENERAL MEETING

Presiding: Omar Legant, M.D., Albuquerque, Immediate Past-President

8:30 a.m. Invocation:
Jack C. Redman, M.D., Albuquerque

Welcome:
Tom Popejoy, Albuquerque, President, University of New Mexico

Welcome:
Eugene P. Szerlip, M.D., Albuquerque, President, Bernalillo County Medical Ass'n.

Presidential Address:
Robert P. Beaudette, M.D.

FIRST CLINICAL SESSION

Presiding: Robert P. Beaudette, M.D.

9:00 a.m. "The Newer Penicillins"
Jay P. Sanford, M.D., Dallas, Professor of Medicine, University of Texas Southwestern Medical School

9:45 a.m. "Prevention of Rubeola and Rubella"
Alexander L. Kisch, M.D., Albuquerque, Assistant Professor of Medicine, University of New Mexico School of Medicine

10:15 a.m. "Urinary Tract Infection"
Jay P. Sanford, M.D.

10:45 a.m. Intermission

11:00 a.m. Panel: "Infections"
Panelists:
Alexander L. Kisch, M.D.
Jay P. Sanford, M.D.

SECOND CLINICAL SESSION

Presiding: Thomas L. Carr, M.D., Albuquerque, President-Elect

2:00 p.m. "Practical Aspects of Oxygen Therapy"
Theodore N. Finley, M.D., Albuquerque, Associate Professor of Medicine, University of New Mexico School of Medicine

2:45 p.m. "Treatment of Respiratory Distress Syndrome"
William H. Tooley, M.D., San Francisco, Associate Professor of Pediatrics and Staff, University of California Medical Center

3:30 p.m. Intermission

3:45 p.m. Panel: "Inhalation Therapy"
Panelists:
Theodore N. Finley, M.D.
William H. Tooley, M.D.

Evening Program
Western Skies Hotel

7:00 p.m. Cocktail Party

8:00 p.m. Dinner-Dance
Introductions
Presentation of Past-President's Pin

Friday, May 13
Student Union Building
University of New Mexico

THIRD CLINICAL SESSION

Presiding:
Emmit M. Jennings, M.D.,
Roswell, Vice-President

9:00 a.m. "Treatment of Shock"
Max H. Weil, M.D., Los Angeles, Associate Professor of Medicine and Director, Shock Research Unit, University of Southern California

9:45 a.m. "Treatment of Burns"
Walter E. Switzer, M.D., Lt. Col., MC, Chief, Clinical Division, U. S. Army Surgical Research Unit, Brooks Army Medical Center, Fort Sam Houston, Texas

10:30 a.m. Intermission

10:45 a.m. Panel: "Shock"
Panelists:
Martin Brandfonbrener, M.D., Albuquerque, Associate Professor of Medicine, University of New Mexico School of Medicine
James S. Clarke, M.D., Albuquerque, Chairman, Department of Surgery, University of New Mexico School of Medicine
Walter E. Switzer, M.D.
Max H. Weil, M.D.

FOURTH CLINICAL SESSION

Presiding: John D. Abrums, M.D.,
Albuquerque, Secretary-Treasurer

2:00 p.m. "Tranquilizers and Energizers"
Leo E. Hollister, M.D., Palo Alto, Associate Chief of Staff, Veterans' Administration Hospital

2:45 p.m. "Birth Control from the Clinician's Standpoint"
Charles C. Bollinger, M.D., Gallup, Chief, Obstetrics and Gynecology, U. S. Public Health Service Indian Hospital

3:30 p.m. Intermission to View Exhibits

3:45 p.m. "The Problem of Contact Lenses"
Philip P. Ellis, M.D., Denver, Associate Professor and Head of Ophthalmology, University of Colorado

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Wm. Beaumont General Hospital Schedules Seminar, April 28-29

The Third Annual William Beaumont General Hospital Medical-Surgical Seminar will be held April 28 and 29, 1966. The Seminar is accredited by the American Academy of General Practice for 12 Hours credit.

Medical Consultants include Dr. William H. Crosby, Boston, Chief of Hematology, New England Medical Center Hospitals; Dr. Arthur Selzer, San Francisco, Director of Cardiopulmonary Laboratory, Presbyterian Medical Center; and Col. Norman Scott, MC, Washington, D.C., Chief of the Gastroenterology Service, Walter Reed General Hospital.

Surgical Consultants include Dr. Victor Richards, San Francisco, Chief of Surgery, Presbyterian Medical Center; Col. John A. Moncrief, MC, Fort Sam Houston, Texas, U.S. Army Surgical Research Unit, Brooke Army Medical Center; and Dr. A. R. Curreri, Madison, Wisconsin, Professor of Surgery, University of Wisconsin.

The program will be varied and include formal papers, panel discussions and problem clinics. The program will open at 8:35 a.m. on the 28th in the Post Theater.

Subjects include Emotional Aspects of Ulcer Disease, Gastrointestinal Manifestations of Hematological Disorders, Choice of Operation for Duodenal Ulcer, Panel Discussion on Post-Gastrectomy Syndrome, Problems in Neoplasia, Gastrointestinal Problem Clinic, Fluid and Electrolyte Problems, Chemotherapy of Cancer, Stress Ulcer, Management of Congestive Heart Failure, Panel Discussion of Hiatus Hernia, General Medical Programs, Chest and Cardiovascular Problem Clinic, and Problems of Trauma and Burns.

Grand Rounds

at William Beaumont General Hospital, *El Paso*

Medical Problems Related to the Hostile Environment

Moderators: Lt. Col. Robert H. Moser, MC,
Chief, Department of Medicine, and
Maj. Frank H. Chamberlin, MC,
Chief Resident in Medicine

I. **Problems Related to Closed Ecologic Systems** —
Capt. Timothy Harris, MC

II. **Problems Related to High Altitude** —
Capt. Martin Cohen, MC

III. **Problems Related to Heat** —
Capt. Frank Ross, MC

IV. **Problems Related to Cold** —
Maj. Thomas C. Birk, MC

I. Problems Related To Closed Ecologic Systems

Capt. Timothy Harris, MC

Ecology is the science of organisms as affected by factors in their environment. Man's constant exploration of the unknown has involved him in many environments which are hostile to his survival. As a result, he often tries to take his natural environment with him. This may be hostile as well.

There are some interesting problems when man tries to take his atmopshere with him or tries to reproduce it during undersea exploration. The advent of scuba apparatus, cheaply and commercially available, makes any small body of water a potential hazard.

The major problems of scuba diving relate to (1) the behavior of gases under pressure and (2) the rather rapid increase in pressure that occurs with even a shallow dive under water. Boyle's law applies to the living organs as well as in a physical laboratory. A dive only 33 feet below the surface causes an increase of one atmosphere pressure on the individual and a corresponding increase in the pressures of gases in the organism. In other words, a person whose lung is only one-half inflated at a depth of 33 feet would, upon suddenly rising to the surface with its attendant decrease in pressure, have a fully inflated lung.

Increasing the pressure will increase the total amount of a gas dissolved in body tissue. Hence blood and tissue on the surface will contain only one-half the amount of dissolved gas that they contain at a depth and pressure of 33 feet of water.

The practical medical aspects of scuba diving are important and relate not only to diving under heavy pressures of water but also to the usage of the hyperbaric oxygen chambers which are now an important aspect of medical therapy.

With descent, the increasing pressure will be

transmitted throughout the individual; however, if a eustachian tube or sinus is plugged, then pressure will not equalize in all chambers and a situation of a relative vacuum exists. Blood vessels will swell and rupture; pain will occur. If the pressure in the face mask is not equalized, a variant of this, "The Squeeze", will occur about the face and eyes. In former days, when helmets did not have a means to equalize pressures, divers were sometimes squeezed into their helmets at great depths.

Once man is on the bottom, he has other problems. As the partial pressure of N_2 increases and more of the gas is dissolved in tissues, the N_2 behaves as an anesthetic (having a 5:1 fat solubility ratio) and a condition called nitrogen narcosis or "rapture of the deep" occurs. The effect is much the same as alcohol and Duffner has dubbed this Martini's Rule: at pressure of 100 feet, the dissolved N_2 acts as one martini, and by 300-400 feet, the effect is that of too many martinis; usually death from disorientation results. While there is much individual variation, the same problem has been observed in surgeons operating in hyperbaric chambers at pressures of 3-5 atmopshere of air, judgment has been impaired. The symptoms are the same.

The composition of the gases used in diving is important. One hundred per cent O_2 will always cause seizures and coma at pressures equal to 33 feet below the surface within 30 minutes. This is equal to 2 atm of pressure. The same effect will occur in the patient given 100 per cent O_2 under a comparable pressure in a hyperbaric tank. Fortunately, most patients are protected by anesthesia and present no problem, but the importance of this fact in an unanesthetized cyanotic congenital cardiac is obvious.

O_2 in moderation is good and vital, but even at 1 atm of pressure over a 24-hour period, 60 per cent of O_2 (O_2 at a pressure of 425 mm Hg) will cause desquamation and congestion of alveolar walls with hyaline-like membrane formation. This becomes a factor in the mixture of gases for swimmers and most important in the gas mixtures and pressures used by astronauts.

The most familiar problem is that of too rapid change from the great pressure of deep water to the surface. This involves three situations, all related to decreasing the pressure on body gases, which may be in a liquid or gas phase.

The air in a lung at 125 feet below the surface will increase five times in volume by the time the surface is reached; slow continued exhalation will solve this. However, a small area of alveoli obstructed by an inspissated plug, or a fragile emphysematous bleb which results in trapped air, may cause serious consequences, ballooning out and rupturing. Interestingly, the body cannot recognize that a ballooning out of thoracic volume is occurring until the rupture.

Coupled with this is the attendant risk of air embolism — e.g., caused by rupturing of this gas into an intrathoracic blood vessel.

The "bends" is the third major result of decreasing the environmental pressure on an individual. Gas dissolved in tissue under high pressures will return to the gas phase as pressure decreases. It will bubble forth in tissue if the pressure decreases faster than the circulatory and the pulmonary system can carry it away. Nitrogen, which is a slowly diffusing gas with a low solubility, is the major culprit. The most serious complications result from the pressure of the bubbles as they occur in closed, inelastic tissue spaces such as brain, spinal cord and some joints. This is a potentially lethal disease, crippling at best, whose only treatment is recompression. The salient clinical features are visual disturbances, CNS manifestations, respiratory distress, skin lesions, and may occur anywhere from 6-24 hours after a dive. To obviate these problems, dive depth is limited and ascent is made in easy, decompressing stages.

The management of gases is even more complex when dealing with prolonged submersion of many people in a submarine. In this instance, the problem of pressure has been negated by the hull. The O_2 source is classified but evidently is inexhaustible and the gases involved are mainly those produced by man and his environment.

The first problem was CO_2 accumulation with its associated toxic effects. A new machine, containing Monoethanolamine (a potent CO_2 absorbent), has been developed which re-uses the amine again and again; however, this is a somewhat volatile hydrocarbon and creates another problem. Apparently, other toxic hydrocarbons occur from paint, waxes, and solvents and carbon monoxide is produced by the crew. A catalytic burner has

been developed to burn all of these compounds. These burners raise another problem. They burn trace elements of Freon (1000 ppm permissible range) which, in themselves, are not harmful, and convert them to HC1, HF1, $C1_2$, $F1_2$, which are highly toxic in small amounts, e.g., 1-2 ppm.

Hence, leak-proof refrigerators and close monitoring of trace gases are required. Even Ozone becomes a problem from electrical arcing (0.1 ppm permissible dose) and if this were not enough, humidity and temperature have to be controlled by a 240 ton unit.

There are many differences between a similar machine designed to support man in a vacuum, namely the space capsule.

The interesting point is that the less pressure within the capsule, the less structural material is needed in the capsule wall. However, man has certain limitations as to the effective pressure he can operate in. The partial pressure cannot be too low or the bends will develop, for going from normal atmospheric pressure to a much lower pressure will have the same effect on our astronaut, as rising to the surface had on the scuba diver. If a person breathes 100 per cent O_2 for a period of two hours, he will "wash out" 75 per cent of the nitrogen in his body. If he then is subjected to a low environmental pressure, the risk of "ebouillon" or bubbling off of nitrogen is markedly lowered. Our astronauts have a pre-flight nitrogen washout with 100 per cent O_2 for two hours. The pressure in the capsule is approximately 5.1 psi or 263.7 mm Hg. They breathe a pure O_2 mixture; notice that the partial pressure of O_2 is lower than our figure of 425 mm Hg which causes pulmonary changes, but is well above the 90 mm Hg mark necessary for Hbg saturation.

In essence, they are atmospherically at a height of 30,000 feet, breathing pure O_2 at a safe pressure with a minimal amount of N_2 in their bodies.

The problems of eliminating noxious gases in a space capsule are the same as in a submarine, limited by weight and room. Lithium hydroxide, a potent CO_2 absorber, is used.

One other problem encountered in the capsule is the effect of or lack of gravity. There are many other parameters but two interesting effects on the cardiovascular system exist.

With acceleration in the range of 5-6 G., an individual reclining and breathing normal air will develop atelectasis and desaturation of blood. Due to pulmonary restriction, secondary to the effect of the transverse forces, his ability to perform simple feats will be impaired. By giving O_2 under pressure, this effect is negated and function is normal.

Finally, weightlessness for 4-6 hours will affect the ability of the cardiovascular system to respond to gravity.

When we stand, blood tends to pool in the lower extremities due to gravity. The cardiovascular system, as a result of the decrease in venous return to the heart, undergoes reflex vasoconstriction to prevent this. Without the effect of gravity for even 4-6 hours, the system loses its ability to respond and upon re-entry, excess blood will pool in the lower extremities and postural hypotension will occur. This was observed mainly in manned Flight 3. Apparently, the problem is one of disuse. If either rotating tourniquets are applied or frequent valsalva maneuvers are performed while at O gravity, thus causing decreased venous return to the heart, reflex vasoconstriction will occur. The mechanism will be kept in condition and will respond promptly to gravity upon re-entry. The astronauts pulled up on a "bungee", a spring-like exercise bar fastened to the floor to provide this exercise. This situation applies to the bedridden patient, for the problem is the same.

It is interesting that as man develops ways of coping with these hostile systems, he also learns more about his friendly ecologic system, Earth.

References

1. Ebersole, J. H.: The New Dimensions of Submarine Medicine, New Eng. J. Med. 262: pp. 599-610.
2. Simons, D. G.: Space Medicine—The Human Body in Space, Monograph #6, Franklyn Institute of Pennsylvania, 1958, pp. 161-178.
3. Duffner, G. J.: Medical Problems Involved In Underwater Compression and Decompression, Ciba Clinical Symposium, Vol. 10, Number 4, pp. 99-114.
4. Giovanni, C. D., and Chambers, R. M.: Project Mercury Report of the Committee on Aeronautical and Space Sciences, United States Senate, Dec. 1, 1959, U.S. Gov. Print. Office.
5. Physiologic and Psychologic Aspects of the Gravity Spectrum, N.E.J.M. 270: pp. 35-40, Jan. 1964.
6. McCally, M., and Graveline, D. E.: Physiologic Aspects of Prolonged Weightlessness, N.E.J.M. 269: pp. 508-516, 1963.
7. Graveline, D. E.: Maintenance of Cardiovascular Adaptability During Prolonged Weightlessness, Aerospace Med. 33:297-302, 1962.

II. Problems Related to High Altitudes

Capt. Martin Cohen, MC

Introduction

One does not have to be reminded of man's legendary and historical enchantment with the charms of space and flying. His developments in air travel since Kitty Hawk in 1903, and his logarithmically-increasing achievements in speed and altitude, reflect his fascination and motivation. However, this has been a relatively unilateral romance with many painful frustrations and rebuffs.

The atmospheric envelope weighs one-millionth of the earth's total weight. It consists of (1) the troposhpere, for the lowest 30,000 feet, with seasonal and geographic variations in temperature, (2) the tropopause, a transition zone at approximately 35,000 feet, and (3) stratosphere, over 35,000 feet, where temperature is -65 to -75F colder over the equator, and constant all year. Above this and into space are the ionosphere and the exosphere. Gaseous composition to 50,000 feet is similar to sea level, with O_2 21 per cent and N_2 78 per cent. Over 50,000 feet, higher concentrations of Ozone appear, reaching peak at 70,000 feet and essentially replacing O_2. Photochemical products of N_2 become significant only over 30,000 feet.[7]

The various problems becoming dominant at graded altitudes are: hypoxia at approximately 10,000 feet, the potential danger of aeroembolism over 30,000 feet, the maximum danger of explosive decompression at approximately 60,000 feet, the increasing Ozone hazard over 60,000 feet, and the progressive increase in cosmic radiation over 70,000 feet.

Hypoxia

The diminshing pO_2 with altitude is a direct result of the fall in barometric pressure, since the gaseous composition of air remains constant. The typical curve relating barometric pressure to altitude demonstrates one-half atmosphere pressure at 18,000 feet, and one-fourth atmosphere at 34,000 feet. The physiologic situation is worsened by the H_2O and CO_2 in the lungs, which displace 47 mm Hg and 30 mm Hg respectively, and which cumulatively reduce alveolar PO_2. Arterial desaturation appears over 7,000 feet; the critical hypoxic altitude is 22,000 feet, at which PO_2 reaches 30 mm Hg and unconsciousness quickly appears. On 100 per cent O_2 at ambient pressures, arterial desaturation first appears at 34,000-35,000 feet, and the critical alveolar PO_2 of 30 mm Hg is reached at 45,000 feet.[7]

Hypoxia is the most dangerous stress of altitude because its effects are insidious, leading to death without clear warning. Because of this treachery, it is recommended that flight crews have practice runs in low PO_2 chambers to teach the individual patterns of reacting to hypoxia. Of course, all the signs are accelerated by any hypoxic pathologic state, e.g., anemia, emphysema, heart disease, and by other stresses, such as fatigue and exertion. Smoking is said to decrease altitude tolerance by 1,000 feet.

Symptoms of hypoxia begin at 5,000-8,000 feet, with decreased night vision and dyspnea on exertion. At 3,000-10,000 feet, fatigue, irritability, increased dyspnea on exertion and headache develop. At 10,000-15,000 feet, decreased judgment, decreased reaction time and further increase in dyspnea on exertion and headache become manifest. At 15,000-22,000 feet, shortness of breath and unconsciousness in 10-30 minutes are the chief symptoms; over 22,000-25,000 feet, unconsciousness develops as a rule in 4-7 minutes. The special senses are affected with diplopia and visual impairment appearing early; hearing is relatively well preserved. Personality changes resembling alcohol intoxication are common and should be predicted.[7]

Recovery with O_2 administration occurs in 10-30 seconds, but a "hangover"-like state may persist. Contributions to symptomatology from the accompanying hypocapnia and respiratory alkalosis are difficult to dissect separately.

The above are manifestations of "subacute" hypoxia, obtained with relatively slow ascent over several minutes at least.

Acute symptoms develop with sudden exposure to low PO_2, as with bailing out or "blow out" of a hatch or window. The appearance time depends on the altitude and the ambient pressure. Over 50,000 feet, sudden decompression leads to rapid

expulsion of alveolar gasses (with rate of expulsion depending on pressure differential), leading to essentially anoxic alveoli almost immediately. Consciousness is maintained until (a) the anoxic blood is circulated from the lung to the CNS (usually 7-10 seconds) and until (b) the brain cell intracellular O_2 is depleted (4-10 seconds). Thus, unconsciousness develops in 10-15 seconds at 50,000 feet; in 2½ minutes at 22,000 feet.

It is to prevent the insidious effects of gradual hypoxia and the virtually immediate effects of sudden anoxia that it is recommended that pilots utilize O_2 masks and/or pressurized cabins over 10,000 feet.

Physiological adjustments to subacute hypoxia begin with increased pulmonary ventilation, to elevate alveolar towards ambient PO_2 as much as possible; this is recognized between 6,000-10,000 feet. The heart rate increases; cardiac output is elevated transiently. After the initial increase in heart rate, cardiac slowing occurs and this may lead to bradycardia collapse. Peripheral capillary vasodilation occurs, presumably to augment tissue flow, but this, too, can lead to collapse with further O_2 deprivation. Pulmonary vascular resistance and pulmonary hypertension correlate best, but nonlinearly, with alveolar/arterial PO_2, with the mechanism being poorly understood.

It is of interest that the myocardium itself tolerates hypoxia quite well, surviving long periods at PO_2 15 mm Hg (equivalent to 40,000 feet); but myocardial function is impaired early, leading to chronic heart failure in 30 minutes. Most episodes of circulatory collapse occur much sooner, almost certainly secondary to reflex action.[6]

Dysbarism

The next problem appearing after hypoxia is decompression sickness, resulting from differences between the barometric pressure and that of the gases trapped in the body cavities or fluids.

Up to 25,000 feet, symptoms are chiefly due to expanding gases in hollow organs. Abdominal distress from gastric distention is common. Pain is said to develop only when volume is two times normal. Flatus and eructation obviously provide relief. Entrapment of air on ascent, or failure of air filling on descent, produces middle ear symptoms. Pain develops when the pressure differential is over 30 mm Hg across the tympanic membrane. Prolonged and repeated disturbances lead to "aerotitis," and similarly to sinusitis. In military pilots on O_2 by mask in high performance, partially pressurized planes, O_2 fills the middle ear cavity at ambient pressures, only to be dissolved several hours later, leading to acute delayed otitis. To limit these minor discomforts, commercial jets attempt to maintain one atmosphere pressure to 22,000 feet with no variations.

Over 25,000 feet, evolution of bubbles on N_2 in blood and tissues leads to symptoms of aeroembolism, the aviator's counterpart of the diver's bends. With gradually increasing altitude, such evolved gases are cleared from tissues via circulation and lungs. With rapid ascent, or more susceptible individuals, particularly the obese, a multitude of symptoms develops as bubbles accumulate. In experimental animals, these first appear in the CSF. Appropriate descriptive terms include the "bends," "chokes," "creeps." Aeroembolism is particularly troublesome to military pilots, as military planes are not highly pressurized to reduce weight.

In commercial planes, decompression sickness is seen only with loss of cabin pressure with altitudes maintained for over 10-15 minutes. The commonest cause is blow-out of hatch or window. The large volume of gas escaping from this small portal requires up to two minutes to decompress. The pilot has ample time to descend at a safe 8,000-12,000 feet/minute to lower, denser altitudes, virtually eliminating the possibility of aeroembolism attacking the commercial passenger.

Another problem of lowered barometric pressure at high altitude revolves about positive pressure breathing. At ambient pressures, 100 per cent O_2 is adequate only to 34,000 feet. Higher than this, positive pressure assistance is required. Profound circulatory alteration develops if the pressure applied to the pulmonary circulation exceeds that on the systemic circulation by over 15-20 mm Hg. Chiefly, there is obstruction to venous return, resulting in decreased cardiac filling and decreased cardiac output. It is said that the Germans rarely used such devices in World War II because of the EKG changes of coronary insufficiency so produced. A further complication of this pulmonary-systemic pressure differential is shift of fluid (by increased venous pressure) to the extravascular space, producing diminished circulating blood vol-

ume. Adequate protection is provided by a tight fitting "G" suit.[6]

Other Problems

Cosmic radiation becomes a problem over 60,-000-70,000 feet. A relatively harmless layer of radiation extends over temperate latitudes at approximately 40,000 feet, averaging only 6 mr/24° — an insignificant exposure. Higher up, at 60,000 feet, increasing radiodensity exposes air crews on average weekly flight tours to accumulated doses of up to 50 R over 10 years. The cosmic radiation consists of heavy primaries, nuclear bodies heavier than proton or alpha particles with great tissue penetrating capacity, at over 90,000 feet; and secondaries, fission products of primaries, at 60,-000-70,000 feet, with little penetrating capacity.[2] It is expected that the time-dilution factor will minimize this problem. A consequential complication is the attachment of fission fragments to portions of high altitude aircraft. These accumulate at areas of high turbulence, mostly turbine blades and leading edges. As yet, this has not become a threat to ground crews who follow basic hygienic rules.[7]

Gravity Forces — "G's"

This is the term applied to the inertial effects of changes of direction, usually at high speeds. The effects depend on the relation of body position to direction of applied force. At sea level, one is subject to "1G," parallel to the spine; impeding flow upwards, aiding flow caudally. The major systemic effects depend on the impact of "G's" on the column of blood between heart and brain. At 1G, this column, approximately 30 cm long, displaces 24 mm Hg; at 2G, 48 mm Hg, and at 4G, 96 mm Hg. This value is essentially subtracted from aortic blood pressure, resulting, at higher "G's," in decreased or absent CNS blood flow. Peripheral vision is lost first, then "gray-out" and blackout. Short, squat individuals with shorter blood columns are more resistant. The average healthy young adult can sustain 3½ G's for several minutes, 5-6G's for under one minute, and extremely high G's for only 2-10 seconds (until brain intracellular O_2 is depleted). The limitation of maneuverability because of this factor is obvious. G-suits, applying lower body pressure proportionally to G's, represent the manmade adaptation to this stress.

Other physical manifestations of G forces include a transient decrease in blood pressure, promptly compensated by vasopressor reflexes; overstress collapse of the heart as the weight of blood at aortic valve proves immovable; transient decreased pulmonary apical blood flow; and loss of intravascular fluid to dependent portions. The tolerance to G's depends on the direction of forces. The above responses are to positive G's, as in sudden climbing. Negative G's, forcing blood to the head, are very poorly tolerated because of detrimental reflexes initiated. Transverse G's, in anterior-posterior direction, are tolerated best, but substitute pulmonary restriction for circulatory insufficiency.[6]

Acute Pulmonary Edema of Altitude

This last topic is a subject of current interest in medicine and cardiology, and concerns the acute pulmonary edema of altitude. It differs from most of the foregoing topics by involving lower altitudes, more prolonged exposure, an element of heavy exertion, and chiefly a civilian population. But it might appear crucially at times in aircraft combat.

This entity is the development in mountain climbers and in natives of mountains returning to heights after some time at sea level of acute pulmonary edema without evidence of heart disease.[4] It usually appears in healthy, active persons at altitudes of 9,000-15,000 feet, after 12-36 hours of exposure. Frequently, unusual exertion in the cold has been undertaken. Several cases in physicians have been reported minutely, first-hand.[3,1] Symptoms consist chiefly of shortness of breath and cough. Findings have confirmed pulmonary edema. Cardiac catheterization studies have revealed pulmonary hypertension, normal wedge pressures, decreased cardiac output, and no evidence of left ventricular failure.[5] The mechanism is poorly understood; it is probably related to reflex precapillary vasoconstriction, although there is controversy over the contribution of pulmonary venular constriction[1] as is implicated in brisket disease of cattle. Improvement has followed return toward sea level and remarkably after O_2 therapy.

In conclusion, this review has examined some of the factors obstructing the relationship between man and one of the objects of his fascination, high altitude. It has considered hypoxia, dysbarism, positive pressure breathing, cosmic radiation, "G" forces, and acute pulmonary edema of altitude. Real progress has been made in this ro-

mance: respect has been engendered; but understanding remains to be furthered before eventual consummation of man's ethereal desires.

References

1. Fred, H. I., Schmidt, A. M., Bates, T., and Hecht, H. H.: Acute Pulmonary Edema of Altitude, Circulation 25: 929, 1962.
2. Graybiel, A.: Future Trends in Military Medicine, Military Medicine 120: 347, 1957.
3. Houston, C. S.: Acute Pulmonary Edema of High Altitude, N.E.J.M. 263: 478, 1960.
4. Hurtado, Alberto: Pathological Aspects of Life at High Altitudes, Military Medicine 117: 272, 1955.
5. Hultgren, H. N., Lopez, C. E., Lundberg, E. and Miller, H.: Physiological Studies of Pulmonary Edema at High Altitude, Circulation 29: 393, 1964.
6. Lamb, L. E.: Influence of Aerospace Flight on the Normal Cardiovascular System — Stresses and Effects, In Lamb, L. E., ed, 1st International Symposium on Cardiology in Aviation, at School of Aviation Medicine, Nov., 1959.
7. Schwichtonberg, A. H., Luft, U. C., and Stratton, K. L.: Altitude Physiology: Air Travel in the Jet Age. In Gordon, ed., Clinical Cardiopulmonary Physiology, 2nd ed., sponsored by American College of Chest Physicians. (New York, Grune and Stratton, 1960). Pp. 936-957.

NOTE: Balance of Grand Rounds to be carried in May issue.

Physicians to Honor Retiring TWC Professor

Physicians from West Texas and Southern New Mexico are invited to attend a dinner honoring Anton H. Berkman, Ph.D., who is retiring from the Faculty of Texas Western College after 39 years of dedicated service and who has been responsible in the course of his career for encouraging several hundred students to enter the field of medicine.

The dinner will be held May 5 at 7 p.m. at the Sheraton-El Paso Motor Inn in El Paso. It is being sponsored by the El Paso County Medical Society, the local chapter of Phi Beta Pi Fraternity, former students of Dr. Berkman, and the Texas Western College Faculty.

Dr. Berkman, born in Round Rock, Texas, has been a member of the Texas Western College Faculty since 1927, when it was the Texas College of Mining and Metallurgy. He received his B.A. and M.A. from the University of Texas, and his Ph.D., in the field of Botany from the University of Chicago.

At Texas Western he has served in many capacities, including Associate Professor and Chairman of the Biological Sciences Department, Acting Dean of Students, Dean of Graduate School, and Dean of Arts and Sciences. At present he is a Professor of Biological Sciences. His official retirement date is June 1, 1966.

Dr. Berkman is an honorary member of Phi Beta Pi and is the founder of the TWC Pre-Med Club. The author of numerous scientific publications, Dr. Berkman has served as Editor for the World Health Organization's Arid Lands Commission. He has conducted research projects for the U.S. Public Health Service and is President of the Southwestern Division of the American Association for the Advancement of Science.

APHORISMS and MEMORABILIA*

(Concluded)

ANDREW M. BABEY, M.D., *Las Cruces, New Mexico*

45. The paradox of the more efficient cholecystographic dyes: as better and better dyes are developed and concentration on the gallbladder becomes more and more efficient, the chances of the dye obscuring gallstones go up and up.

46. Of all excuses one hears for removal of the gallbladder which contains a large solitary stone, that of the danger of cancer is the weakest. Gallbladder is one of the rarer gastro-intestinal sites for cancer to begin. (pg. 85)

47. It is an interesting fact that the jaundice of common-duct cancer is more likely to fluctuate in its intensity than the jaundice of common-duct stones. Never exclude tumor obstruction just because the jaundice improves or even disappears. (pg. 85)

48. Emotional disturbances are so common in patients with chronic pancreatic disease that a pancreatic problem must be suspected in anyone with unexplained abdominal pain and any psychic disturbance. (pg. 86)

49. Within a year of acute pancreatitis, 20 per cent of the patients in some series have developed pancreatic calcification (Arch. Int. Med., *81,* 301, 1948). (pg. 87)

50. Normal gallbladder is present in only about 30 per cent of patients with acute pancreatitis (Surgery, *31,* 614, 1952). (pg. 87)

51. Any obscure acute abdominal problem in which left pleural effusion develops may be a pancreatitis problem. (pg. 87)

52. Carcinoma of pancreatic head: in only about 20 per cent of cases is the jaundice painless (in spite of the classical teaching). (pg. 88)

53. Given the precise size, shape, and composition of a swallowed foreign body, it is possible to make a reliable estimation of the probable rate of passage or to judge the potential risk of damage. (pg. 90)

54. About 65 per cent of women in the second half of pregnancy have esophageal varices. (pg. 95)

55. Of 1001 patients proved to have urinary calculus, 179 had been diagnosed clinically as cholecystitis or ulcer, 138 had only hypogastric pain, 37 were subjected to appendectomy (Surg., Gyn. & Obst., *50,* 106, 1930). (pg. 95)

56. Of various cardiac diseases which may clinically simulate acute abdominal conditions, be particularly alert for acute pericarditis. (pg. 97)

57. Esophageal varices are common and commonly severe in chronic congestive heart failure. (pg. 97)

58. When a person has reached old age, he has demonstrated that he is well endowed with all the protective mechanisms which Nature is able to supply. The young person is untried in this respect. Have faith that your very old patient will withstand a major surgical procedure, if it is indicated. Don't withhold necessary surgery just because of the patient's age. Most do as well as the young person and many do better. (pg. 100)

59. Enema-tip perforation with the deposition of enema fluid pararectally continues to be an occasional and often fatal accident. There is no reason to insert an enema tip beyond the anal sphincter. (pg. 104)

60. As doctors, we all have a make-up which seems super-sensitive to the threat implied in the possibility that organic disease may be overlooked. In particular, we have a fearful objective cancerphobia. Functional gastro-intestinal disease is ordinarily easy to recognize, and the diagnosis must not engender insecurity in the doctor. (pg. 106)

*From: "Pearls in Gastroenterology" by Colonel Eddy Palmer, M.C., USA, Washington, D.C. 1955. Reprinted with permission of Colonel Palmer.

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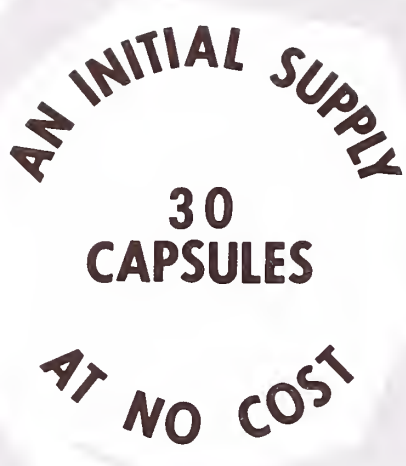

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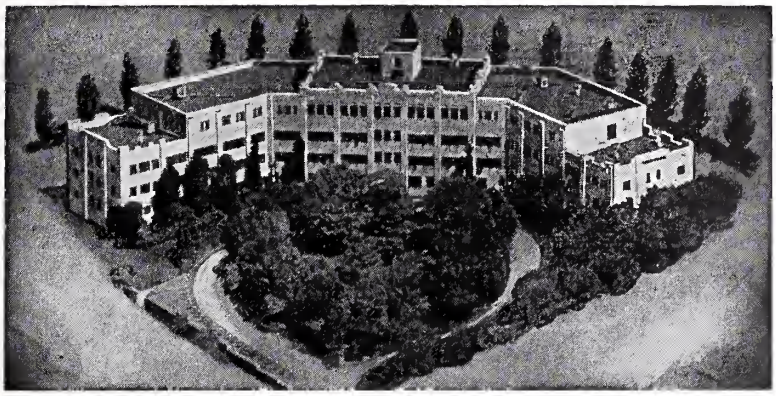

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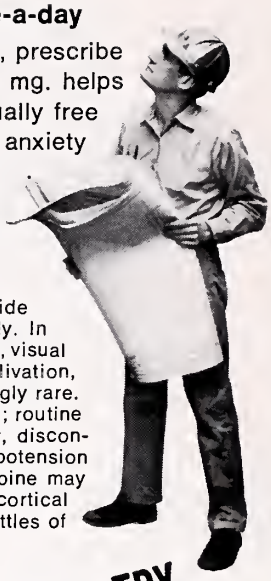

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VOL. 47, NO. 5

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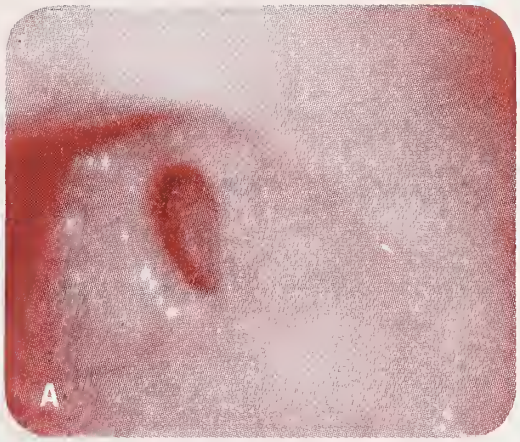

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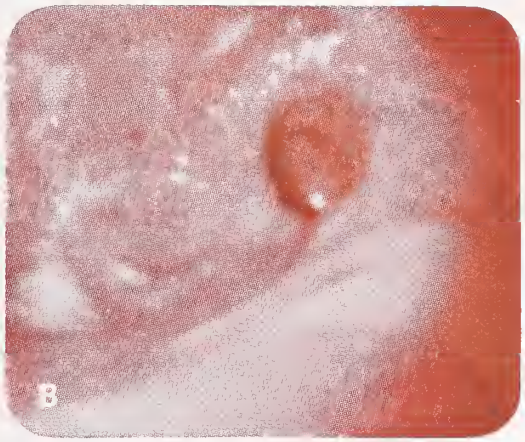

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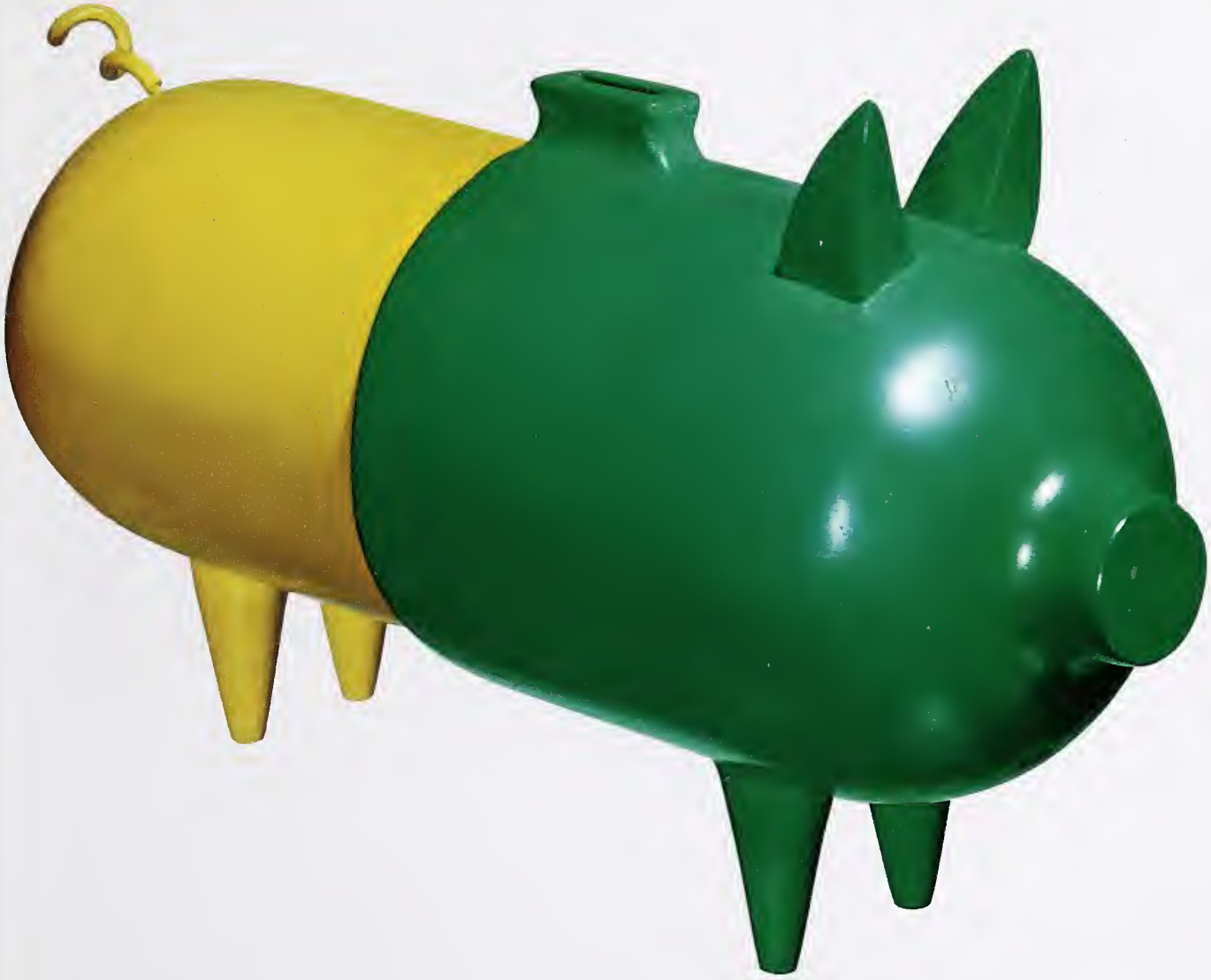

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REFERENCES: 1. Vollmer, H.: Arch. Neurol. and Psychiat., 43:1057, 1940. 2. Morrissey, J.H.: J. Urology, 57:635, 1947. 3. Krantz, J.C., Jr., and Carr, C.J.: Pharmacological Principles of Medical Practice, 2nd ed., Baltimore (1954), 552.

*This one at Westover, elegant Colonial Virginia plantation, located on the James River near Richmond. Built in the early 1730's by William Byrd II, founder of Richmond, it is now the home of Mrs. Bruce Crane Fisher.

A. H. ROBINS COMPANY, INC., RICHMOND, VA.

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Medical Problems Related to the Hostile Environment

(Continued)

Moderators: Lt. Col. Robert H. Moser, MC,
Chief, Department of Medicine, and
Maj. Frank H. Chamberlin, MC,
Chief Resident in Medicine

I. Problems Related to Closed Ecologic Systems —
Capt. Timothy Harris, MC

II. Problems Related to High Altitude —
Capt. Martin Cohen, MC

III. Problems Related to Heat —
Capt. Frank Ross, MC

IV. Problems Related to Cold —
Maj. Thomas C. Birk, MC

III. Problems Related to Heat

Capt. Frank Ross, MC

Historically, the first experiments in disorders of heat regulation in humans were made in 1776 by Dr. George Fordyce and Dr. Blagden, who exposed themselves to dry and moist heat in a specially prepared room. They found that they could remain in dry temperature at 260F for 13 minutes without apparent discomfort or rise in body temperature while a "beefsteak was well cooked and eggs roasted hard." However, similar exposure to moist temperature at 130F caused elevation of body temperature and marked discomfort. Their conclusion was that the moisture of the surrounding air was the principal determining factor causing disorders of heat regulation, by preventing rapid evaporation of sweat necessary for cooling the body at environmental temperatures in excess of body temperature. Their conclusions have been substantiated to a great extent by more recent and more elaborate experiments.

Unfortunately, these recent and elaborate experiments have not unraveled many of the mysteries of the basic pathophysiology of human disorders of heat regulation, primarily because the physiology of the normal heat regulatory mechanisms is not fully understood.

According to Bard's Textbook of Physiology,[1] there are four fundamental mechanisms of heat loss. They are: (1) Conduction — a process in which heat is transferred through solid, liquid or gas from one molecule to another at a rate dependent on the thermal conductivity of the substance and the difference in temperature between the two points under consideration. This means of heat loss is negligible in human heat regulation, primarily because air is such a poor conductor of heat. (2) Convection — a process limited to liquids and gases in which, by the mechanical process of mixing, cooler fluid comes into contact with warmer fluid with subsequent transfer of heat from the warmer to the cooler fluid by conduction. The newly-heated fluid carries the heat elsewhere because its density is less than the cooler fluid around it. (3) Radiation — a process by which heat liberated by the metabolic processes in the body is carried as electromagnetic waves through the air to cooler objects in its environment. The amount of radiation from an object such as the human body is proportional to its surface area, to its emissive power, and to the difference in temperature between the radiating body and the body to which it is radiating. This last fact is

very important in human heat regulation because it means that under normal basal conditions (surrounding temperature less than body temperature), radiation accounts for approximately 40 per cent of heat loss from the human body, while under conditions of high environmental temperature (surrounding temperature greater than body temperature), this means of heat loss is inoperable. In fact, when environmental temperature is greater than body temperature, there is a net gain of heat by the human body under these conditions via the process of radiation.

Convection also contributes approximately 40 per cent to heat loss under basal conditions, leaving approximately 20 per cent for the fourth process — evaporation. However, when environmental temperature is greater than body temperature, convection also is inoperable for heat loss, because the surrounding air is now the warmer fluid and it will transfer heat by convection to the liquid on the body surface rather than remove it. Therefore, at high environmental temperatures, heat is gained — not lost — from the environment by the process of convection. Thus, at high environmental temperatures, the burden of heat loss falls entirely on the fourth process — that of evaporation. (4) Evaporation — a process, wherein a specific amount of heat is utilized to change any liquid to its vapor form without changing its temperature.

Bard concludes, "When a body is at a temperature below that of its surroundings, it can lose heat only by evaporation; at the same time it will be gaining some heat by the other forms of thermal transfer (radiation, convection and conduction)."

The human body loses heat primarily through the skin. The respiratory tract is also used, while heat loss via urine and feces is negligible. The factors determining heat loss are, primarily: (1) temperature of skin and surrounding air, (2) amount of water vapor in air, and (3) movement of air over skin.

The clinical disorders of heat regulation are usually caused by an unfavorable state of one or more of these factors — that is, by (1) severe environmental temperature greater than body temperature, (2) moderate environmental temperature with high humidity, or (3) lack of circulation of surrounding air.

The clinical disorders of heat regulation are classified as: (1) heat cramps, (2) heat exhaustion, (3) anhydrotic heat exhaustion (a new classification recently adopted by the World Health Organization describing a condition somewhat between heat exhaustion and heatstroke), and (4) heatstroke. Although these four categories of disorders of heat regulation are clinically distinguishable, they are probably pathophysiologically a spectrum of one basic disorder in which the heat-regulatory mechanism is respectively more severely deranged and thus the clinical picture more disturbing.

1. Heat Cramps — a condition characterized by severe spasms of voluntary muscles, especially those located in the lower extremities and the abdomen. This condition primarily affects workers performing a great deal of physical exercise in a warm, humid climate. It does not require direct sun exposure or environmental temperature in excess of body temperature. The basic difficulty is salt loss via excessive sweating, with inadequate replacement. The treatment is oral administration of salt, either in tablets or added to drinking water. The condition is not considered serious. Prevention can be accomplished by adding salt to drinking water to a concentration of 0.1 per cent.

2. Heat Exhaustion — a condition characterized by profuse perspiration, cool, clammy skin, low blood pressure, weak thready pulse and symptoms of headache, mental confusion, vertigo, weakness, anorexia, nausea, vomiting and visual disturbances. Occasionally, muscle cramps are seen. The patient is usually conscious at all times. The mouth temperature may vary from subnormal to slightly elevated, while the rectal temperature is normal to slightly elevated.

Darling, in Cecil and Loeb's Textbook of Medicine,[2] describes heat exhaustion as a physiologic breakdown following prolonged exposure to heat. The degree of temperature elevation, the humidity and the wind velocity are important factors. Dehydration and salt deficiency predispose one to its development. So do the extremes of age (childhood, old age), acute or chronic disease, and strenuous physical exercise. Pathophysiologically, one initially finds a marked increase in skin and muscle circulation associated with inadequate cardiac output, due to decrease in circulating blood volume and disabled vasomotor control. The circulatory blood volume is decreased because of the large volumes of water and salt lost via the evaporation of sweat. This condition occurs primarily when a person has not been acclimatized. Acclimatization is a physiologic process that occurs

over a 4-7 day period of gradually increasing work expenditure in warm environment, whereby the output of aldosterone increases so that there is a decrease in urine and sweat content of salt and a subsequent increase in blood volume. Subsequently, the phenomenon of "escape" occurs in which urinary sodium and chloride increase but salt is still conserved via marked decrease in sweat content of salt to negligible levels.

The disabled vasomotor control seen in heat exhaustion is felt by some to be due to the histamine and/or histedine in sweat. This produces peripheral vasodilatation in an effort to increase heat loss via radiation and conduction, but eventually causes peripheral vascular collapse with decreased venous return further compromising the cardiac output.

Heat exhaustion can be prevented primarily by decreasing the duration of exposure to high environmental temperature by decreasing the degree of physical exercise in this environment, and by increasing salt and water replacement.

Treatment consists of removal of the person to a cool, well-aerated area and administering oral fluids (with salt), if possible. If this is contraindicated because of nausea or vomiting, then IV saline may be administered. In very serious cases (very rare), blood or plasma expanders may be necessary. The extremities should be massaged to increase venous return. The prognosis is good and recovery complete. With recovery, there is no greater predisposition to future attacks of heat exhaustion.

3. Anhydrotic Heat Exhaustion — a condition characterized by moderately elevated body temperature (99.2-102F), associated with dry skin and symptoms similar to heat exhaustion. The lack of perspiration differentiates this from ordinary heat exhaustion. This condition generally occurs when the environmental temperature is moderate (less than body temperature), but the humidity is very high. It is felt that the high humidity predisposes to exhaustion of the sweat glands, but heatstroke does not develop because the environmental temperature is less than the body temperature, allowing heat loss via radiation, convection and vaporization of water from the respiratory tract. Prevention and treatment are similar to those of heat exhaustion, described previously.

4. Heatstroke — unlike the previous clinical entities, this condition is very serious; in fact, it is considered a medical emergency of the first magnitude. It occurs in the civilian population, primarily in the elderly, in the obese, during acute alcoholic states, and in people with chronic diseases.

However, the majority of reported cases come from the military services. During WW II, there were over 200 deaths from heatstroke in Marine Corps recruits. Shickele, reporting in Military Surgeon (1947) describes 157 cases in Army trainees. The highest risk was in the obese, unseasoned recruit from a temperate climate, training in a warm, humid environment without proper acclimatization. With the institution of better acclimatization programs, the incidence of this disorder in the period 1956-1960 dropped to one-tenth of the 1950-1954 level.

Heatstroke is a condition in which a person exposed to elevated environmental temperature (usually in excess of body temperature), especially when the humidity is high, suddenly ceases to sweat. Since evaporation ceases, the body has lost its only mechanism for heat loss, and body temperature rises rapidly as the body absorbs heat by radiation, convection and conduction. The person develops headache, mental confusion, staggering gait, delirium, and finally, coma, usually with convulsions. This agitated state increases internal heat production, which further aggravates the rising body temperature. The skin is hot, red and dry. The pulse is rapid and full. The blood pressure initially is normal or elevated. Petechial hemorrhages are seen on the skin and subconjunctival surfaces. The blood shows a leukocytosis and often an elevated BUN. The urine contains albumin and hyaline casts. The EKG shows sinus tachycardia, S-T segment depression, and frequently T-wave inversion. If one is not aware of the environmental exposure, the differential diagnosis is usually with central nervous system disease, sepsis, delirium tremens, terminal liver disease, or thyroid storm.

The prognosis of untreated cases is very poor with almost 100 per cent mortality. With treatment, the mortality varies from 10-90 per cent, depending on the general health of the individual, the duration of hyperpyrexia before treatment, and the degree of hyperpyrexia. Temperature in excess of 106F is not uncommon and is a poor prognostic sign. The initial 24 hours are most important. If the patient survives this period, recovery frequently occurs. However, there may remain residual damage, especially to the central nervous system, the myocardium, the liver and the kidney.

The pathophysiology of this entity is unclear. The etiology of the exhaustion of the sweat mechanism is unknown. There are a number of theories based on experimental and clinical studies.

I. Hypothalamic Disturbance — It has been noted frequently that the hyperpyrexia commonly seen with hypothalamic lesions has been accompanied by a lack of sweating. This hyperpyrexia and lack of sweating is felt to be due to disturbance of anterior hypothalamic centers concerned with temperature regulation via increased sweating and peripheral vasodilatation. This commonly occurs when craniopharyngiomas are removed surgically, and it has been noted that cortisone will frequently prevent the development of hyperpyrexia and the lack of sweating seen post-operatively. This has been one of the arguments offered for the use of steroids in heatstroke. However, as stated by Kahn (1963),[4] in hypothalamic hyperpyrexia, there is usually peripheral vasoconstriction with cool skin as opposed to heatstroke where there is peripheral vasodilatation and warm skin. Steroids are known to act synergistically with norepinephrine to cause peripheral vasodilatation and splanchnic vasoconstriction. Since these are already present in heatstroke and contributing to circulatory distress, the addition of steroids may be unwise in heatstroke.

II. High Output Cardiac Failure — According to Gold (JAMA 1960),[5] in experiments with human subjects, there is a high output cardiac failure in heatstroke due to: (1) marked peripheral vasodilatation with functional A-V shunting leading to marked increase in venous return, with subsequent increase in cardiac output; (2) failure of the left ventricle to keep pace with the increased flow from the right side because of subendocardial hemorrhages and/or infarcts commonly seen in heatstroke. As high output failure proceeds, the venous pressure rises. Gold feels it is the increase in venous pressure that causes the cessation of the sweat mechanism. As evidence, he cites the decreased sweating in cardiac failure (found by Burch) to 25-50 per cent of normal values in hot and humid climates. However, Daily and Harrison (1948)[6] were unable to find increased venous pressure until the terminal stage in experimentally-induced hyperpyrexia in rats. Ferris et al[7] also found normal venous pressure in the human form of the disease.

III. Peripheral Vascular Collapse — Daily and Harrison,[6] in their experiments on rats, found that hyperpyrexia and coma developed as the splanchnic bed dilated (which they felt was due to damage to the vasomotor centers in the central nervous system). Venous pooling ensued, with decrease in the cardiac output and shock. They found no pulmonary edema in their rats unless they were treated with saline infusions. Other experimenters feel the peripheral vascular collapse is primarily due to the extravasation of fluid from the vascular tree due to endothelial wall damage caused by the hyperpyrexia.

IV. Primary Sweat Gland Exhaustion — Thaysen and Schwartz (1955)[8] cite experimental evidence for primary fatigue of the sweat glands. They showed that repeated injections of Mecholyl intradermally will cause decreasing amounts of sweating response in that area because of exhaustion. Generalized heating thereafter caused a significantly greater response in sweat output in areas not pre-treated with Mecholyl. Also, intradermal injection of atropine in one arm during generalized heating would cause inactivity of the sweat apparatus; subsequent generalized heating would cause a significantly greater response in the arm in which the sweat glands had been "rested" by the atropine injections. Wolkin[9] also presented eight patients in which anhydrosis of the entire body, except the face and neck, occurred on exposure to excessive heat. He postulated the excessive heat had a direct effect on some sweat glands, with them subsequent fatigue, whereas other glands were resistant. He felt the persistent face and neck perspiration prevented the development of heatstroke.

Treatment

Heatstroke is a medical emergency requiring immediate treatment. The best treatment is immediate immersion in an ice water bath until the body temperature is less than 102F. Then the patient should be removed and his temperature followed closely. It may drop to normal or subnormal levels at which time the patient should be covered with blankets in order to prevent circulatory collapse.

However, more commonly, the body temperature will rise again. If this occurs, wet sheets should be placed around the patient and electric fans used to increase evaporation. The body temperature must be watched closely for the first 24 hours. Thereafter, the body temperature may remain stable. If ice water baths are not immediately available, then covering the body with cool water and fanning should be started until proper

facilities are found for ice water bath. Ice water baths have theoretic disadvantages such as (1) peripheral vasoconstriction, with decreased heat loss, and (2) predisposition to vascular collapse. In fact, Hoagland and Bishop (1961)[10] state that ice water baths are unphysiologic and may precipitate shock in these patients, and should not be used. However, in spite of these theoretical disadvantages, ice water bath is still the primary treatment of these patients because it is the fastest method of lowering the severe hyperpyrexia which is felt to be the primary cause of the marked cellular damage seen pathologically.

In addition to ice water baths, massage of the extremities to increase venous return of cooled blood from the peripheral vascular tree should be performed.

IV saline should be given, but slowly and in moderate amounts to prevent pulmonary edema. Blood or plasma expanders should be reserved for very severe cases that have progressed to shock.

Steroids are often given, although there is no good rationale for their use.

Chlorpromazine is given by many for the control of the hyperactive state in order to decrease the metabolic load. However, Hoagland and Bishop[10] recommend their use on other grounds. They cite the work of Benette and Laborit in the German Pharmacological Society meetings of 1953, showing that phenothiazines have a hypothermic action, probably via a central mechanism, as well as by decreasing the metabolic rate and oxygen consumption, and also help prevent shock in animals from various causes. They recommend using Chlorpromazine IV, 50 mg every half hour, plus Dypyrone ("Novaldin"), an antipyretic, in place of ice water baths, as a more physiologic means of treating heatstroke. However, this method has not gained many followers in the literature since it was first described in 1961.

Gold[5] recommends rapid digitalization in order to reduce the elevated venous pressure which he feels is the primary cause of the cessation of sweating.

Barbiturates and Morphine should be avoided, because they disturb heat regulatory mechanisms.

Complications of Heatstroke

Pathologically, the primary targets of heatstroke are: (1) the central nervous system with the cerebrum and cerebellum being primarily affected. Edema occurs initially, followed later by nerve cell damage and death, especially of the Purkinje fibers, with secondary gliosis. Recurrent mental confusion, during the first few days of therapy, is not uncommon. Residual cerebellar dysfunction occurs not infrequently. The hypothalamus often appears normal histologically. (2) The heart — subendocardial hemorrhages occur, especially on the left ventricular wall of the intraventricular system. Myocardial infarction without coronary disease is seen. (3) Kidney — Acute tubular necrosis, secondary to the shock-like state, is seen in 2-9 per cent of cases. Hyperkalemia, secondary to marked cellular damage and renal failure, is not infrequently a cause of death. (4) The liver — Central lobular necrosis and/or bile stasis occurs. This is usually reversible. (5) Hematopoietic system — Purpura commonly seen in the early stages is probably on a vascular basis. Thrombocytopenia develops sometimes, often with decrease in Megakaryocytes in the bone marrow. Also, prothrombin production by the liver may decrease. Increased fibrinolysis, secondary to release of tissue activators of plasminogen system, has been described as well as hypofibrinogenemia, probably secondary to decreased liver production. All of these factors make bleeding a very important complication.

Prevention

Prevention is vital because of the gravity of this condition, with mortality at least 40 per cent in severe cases even with proper therapy. It should also be remembered that once a person has had heatstroke he has much greater risk to have recurrent attacks because of residual damage to the heat regulatory mechanisms.

Prevention consists of:

I. Slow "acclimatization" of recruits over a two week period with graded, progressive increases in work load and duration, especially under high environmental temperature. The hardest work should be reserved for the coolest time of the day. Frequent rest periods are essential, during which time adequate fluid replacement with "salted" water should be encouraged. At night, barracks should be cool and dry to allow heat regulatory mechanisms to "rest."

II. Decreased length of periods of exposure to very high temperatures, especially when humid. In this regard, the use of the "wet bulb globe temperature index of Yaglon" as the indication of heat stress has been very important in reducing the incidence of heat disorders. This instrument reflects the combined factors of temperature, humidity, wind velocity and radiant energy from the sun. When the index is between 80-85F, work

loads for young recruits should be decreased, and rest periods increased. Between 85-88F, all training of unacclimatized recruits should be withheld, if possible. Above 88F, even acclimatized individuals may suffer adverse effects from the environment and it is wise to interrupt all strenuous physical activities temporarily.

III. Proper clothing. A minimum amount of loose-fitting, light garments should be worn.

IV. Special regard for high risk cases, obese persons and persons from temperate climates, training in warm, humid climates, must be given.

In summary, I would say that in disorders of heat regulation, one should remember the proverb, "An ounce of prevention is worth a pound of cure."

References

1. Medical Physiology, 11th ed., Philip Bard, ed., St. Louis: C. V. Mosby, 1961, pp. 532-540.
2. Textbook of Medicine, 11th ed., Cecil-Loeb, W. B. Saunders Co., 1963, pp. 1808-1810.
3. Skickele, Elizabeth: Environment and Fatal Heat Stroke, The Military Surgeon, Vol. 100-101, 1947, pp. 235-254.
4. Kahn, E. A.: Heat Stroke and Temperature Control, The Journal of the Michigan Medical Society, 62:292-6, (March) 1953.
5. Gold, Joseph, Capt., (MC) USAF: Development of Heat Pyrexia, JAMA 173:1175, 1960.
6. Daily, W., and Harrison, T.: A Study of the Mechanism and Treatment of Experimental Heat Pyrexia, Am. J. Med. Serv., 215:42, 1948.
7. Ferris, E. B., Jr., et al.: Heat Stroke, Clinical and Chemical Observations on 44 Cases, J. Clin. Invest., 17:249-62, (May) 1938.
8. Thaipen, J., and Schwartz, I.: Fatigue of the Sweat Glands, J. Clin. Invest., 34:1719, 1955.
9. Walker, J., Capt.; Goodman, J., Capt.; and Kelly, W., Capt., (MC) USA: Failure of the Sweat Mechanism in the Desert, JAMA, 124:478, 1944.
10. Hoagland, R. J., Col., (MC), and Bishop, R. H., Jr., Capt., (MC): A Physiologic Treatment of Heat Stroke, Am. J. Med. Serv., Vol. 241, #4, pp. 43/415, (April) 1961.

IV. Problems Related to Cold

Maj. Thomas C. Birk, MC

Cold injuries have been of primary concern to the military forces because of their importance in many campaigns, such as Napoleon's in Poland, and Hitler's in Russia. In World War II in the U. S. Army, there was a total incidence of 90,535 time-lost injuries due to cold, and in the Korean War, there were more than 9,000 such cases.[11] Cold injuries occur only sporadically among the civilian population as in one report from Detroit in January, 1963 when 31 patients were treated for frost-bitten ears after only 15-30 minutes' exposure to -11F,[4] but they remain a problem.

The type of injury produced is divided into four categories: chilblains, immersion foot, trench foot, and frost bite. Chilblains result from exposure to temperature above freezing in high humidity and produce a painful erythema which clears rapidly on warming. Immersion foot results from exposure for from 12 hours to seven days, to water at temperatures usually below 50F. Trench foot results from prolonged exposure to cold and wetness at 20-50F. Frostbite is actual crystallization of tissue fluids in the skin or subcutaneous tissues, occuring after exposure of only a few seconds to several hours, at below freezing temperatures.[9,11]

Cold is the main factor producing cold injuries, but this is enhanced by wind or moisture, which removes skin heat by conduction, radiation and convection.[3] Children and elderly people are the most susceptible; while, in the military, the lower ranks experience the most cold injuries. Previous cold injuries, fatigue, training, discipline, nutrition, health and psychosociological factors all play a role in producing cold injuries. The Negro is approximately six times more vulnerable than the Caucasian, and his injury is usually more severe.[11]

The pathogenesis of tissue injury resulting from cold is not clearly understood, but four mechanisms are thought to contribute:[11]

(1) Direct metabolic impairment due to cold. Degenerative changes in muscle fibers occur after 30 minutes of freezing, while ischemia of the same duration produces no injury; therefore, it is felt that the injury is not due to ischemia alone. Cold, per se, has been shown to affect certain cellular enzyme activities, and denatures certain lipoproteins affecting nerve tissue.

(2) Vascular damage, with decreased tissue perfusion and tissue hypoxia. Corpuscular clumping, capillary stasis, irreversible occlusion of small vessels, and increased capillary permeability all occur. There is also A-V shunting proximal to extremity injury.

(3) Cellular structural damage due to the mechanical effect of freezing. This is probably due to rupture of intercellular bridges and mechanical distortion of organized tissue.

(4) Intracellular molecular changes due to hyperosmolarity subsequent to crystallization of intracellular water. Severe cellular dehydration occurs causing protein denaturation.

Cold injuries often have an insidious onset with a tingling or mild aching sensation being the only symptoms, followed by numbness. An attempt has been made to clinically classify cold injuries as has been done with burns. Unfortunately, this classification becomes evident only after the injury has manifested its outcome.

(1) First degree — Hyperemia and edema. After warming, the skin becomes red, hot and dry, and edema develops within three hours. The

edema clears in about five days, then desquamation of the superficial layers of the skin occurs. Healing is usually complete without sequelae.

(2) Second degree — Hyperemia and vesicle formation. Blisters and huge blebs appear within 6-12 hours. There is usually deep, aching pain associated with intense burning requiring analgesics. The vesicles dry, forming black eschars which gradually separate, revealing intact skin.

(3) Third degree — Necrosis of skin and subcutaneous tissue. There is much edema, vesicle formation and pain. When the eschar separates, there is poorly-vascularized granulation tissue which gradually epithelializes. Secondary infections frequently complicate recovery. Healing occurs in an average of 68 days and there may be prolonged post-frostbite symptoms of hyperhidrosis and cyanosis.

(4) Fourth degree — Complete necrosis and loss of tissue. There is destruction of the entire thickness of the part, including bone, resulting in loss of the injured part. Dry gangrene and mummification occur. A demarcation line appears in about 36 days and extends down to the bone in 60-80 days.

The treatment of cold injuries can be divided into emergency first aid treatment and subsequent hospital care. If the patient has to ambulate after injury, thawing should be delayed.[8] If he can be evacuated by litter, then rapid thawing should be performed as soon as possible by placing the frostbitten extremity in a constant-temperature water bath at 40C (104F).[5] The use of snow, rubbing of the extremity, various ointments, ingestion of alcohol, and tobacco is to be avoided.[10,11] After thawing, a dry fluff dressing is applied and tetanus toxoid administered. Antibiotics should be used if there is the slightest possibility of infection. In the hospital, the patient is placed on absolute bedrest with the extremity exposed to warm room temperature with sterile precautions employed. Whirlpool baths are used twice daily for gentle debridement and cleansing. Physiotherapy should be used to prevent ankylosis of the joints.[7] Surgical debridement or amputation should be avoided and physiologic amputation allowed to occur. There has been much controversy in the past regarding regional sympathectomy for the involved extremity, but recent studies seem to support its merits, especially when performed in the 36-72 hours following injury.[12] Late sympathectomy has also been used successfully when post-frostbite symptoms are particularly severe. Steroids and vasodilators are not indicated. Anticoagulants are probably of value if used within a few hours of the injury, but this is often not practicable. Recently, the use of low-molecular weight dextran to inhibit intravascular cellular aggregation, decrease blood viscosity, and improve blood flow has been advocated, but this has not been fully evaluated.[11]

General hypothermia is treated by placing the patient under warm blankets in a warm room. Rapid re-warming in a bath should be avoided as it may produce shock; and cardiac arrhythmias occur frequently, requiring continuous observation of the patient and vigorous treatment.

Obviously, the best treatment of cold injuries is prevention. This requires a continuous education program from people exposed to cold. Adequate, protective, dry, clean clothing of many layers is most useful. Almost all cold injuries can be prevented if the proper precautions are taken.

References

1. Golding, M. R., de Jong, Pieter, Sawyer, P. N., Hennigar, G. R., and Wesolowski, S. A.: Protection From Early and Late Sequelae of Frostbite by Regional Sympathectomy: Mechanism of Cold Sensitivity Following Frostbite, Surgery 53: 303-308, 1963.
2. Golding, M. R., Mendoza, M. F., Hennigar, G. R., Fries, C. C., and Wesolowski, S. A.: On Settling the Controversy on the Benefit of Sympathectomy for Frostbite, Surgery 56: 221-231, 1964.
3. Hall, W. H.: Medical Problems of Military Operations in Extremes of Heat and Cold, Milit. Med. 129: 641-651, 1964.
4. Johnston, J. Peter: Frostbitten Ears, Henry Ford Hospital Med. Bull. 11: 453, 1963.
5. Pichotka, J., and Lewis, R. B.: Effect of Rapid and Prolonged Rewarming on Local Cold Injury, U. S. Armed Forces Med. J., Vol. II: 1293-1310, 1951.
6. Shumaker, H. B., and Kilman, J. W.: Sympathectomy in the Treatment of Frostbite, Arch. of Surg. 89: 575-585, 1964.
7. Simeone, Fiorindo: Cold Injury, Arch. of Surg. 80: 396-405, 1960.
8. Washburn, Bradford: Frostbite, N.E.J.M. 266: 974-989, May 10, 1962.
9. Yarington, C. T.: The Local Cold Injury, Rocky Mountain Med. J. 58: 27, 1961.
10. Yarington, C. T., and Bales, G. A.: The Local Cold Injury: With Special Reference to Frostbite of the Ears, Milit. Med. 128: 1024-1026, 1963.
11. Departments of the Army, Navy, and the Air Force: Cold Injury, p. 16, Government Printing Office (TB MED 81), Washington, D. C., Oct. 20, 1964.

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Ruidoso Clinic to Hold Joint Meet with ACOG

The New Mexico Chapters of the American Academy of General Practice and the American College of Obstetrics and Gynecology will hold a joint meeting at the Ninth Annual Ruidoso Summer Clinic in Ruidoso, New Mexico. The AAGP dates are from July 25 through 28, 1966, and the ACOG dates, July 29 and 30.

The Faculty for the Ruidoso Clinic will be provided by the University of New Mexico School of Medicine. The meeting is sponsored by the New Mexico Chapter of the American Academy of General Practice. The joint meeting, with six days of attendance for morning sessions, will earn attending physicians a total of 22 hours in Category I credit.

Speakers for the Ruidoso meeting from the University of New Mexico School of Medicine will include Dr. Martin Brandfonbrener, Associate Professor of Medicine; Dr. Frederick Cohn, Adjunct Instructor of Obstetrics and Gynecology; Dr. Arnold H. Greenhouse, Associate Professor of Medicine; Dr. William R. Hardy, Assistant Professor of Medicine; Dr. Alexander L. Kisch, Assistant Professor of Medicine; Dr. Eugene L. Klingler, Jr., Instructor in Medicine; Dr. John K. Leach, Assistant Professor of Medicine; Dr. William S. Lovekin, Assistant Professor of Medicine; Dr. Robert A. Munsick, Professor of Obstetrics and Gynecology; Dr. Solomon Papper, Professor of Medicine; Dr. Carlos A. Vaamonde, Instructor in Medicine; and Dr. Robert Whang, Assistant Professor of Medicine.

Among the topics to be discussed at the Ruidoso Clinic are "Evaluation of a Patient with Renal Calculi," "Practical Aspects of the Diagnosis of Acute Renal Failure," "Technique and Indications for Peritoneal Dialysis," "The Nephrotic Syndrome," "The Indications for Cardiac Catherization," "Drugs and Hypertension," "Diet and Other Factors Influencing Coronary Disease," "Current Concepts in Cerebral Vascular Disease," "The 'Pill' as Related to Contraception and Menopause," "Hypofibrinogenimea in Pregnancy," "Urinary Tract Infection in Pregnancy," and "Normal and Abnormal Labor."

In the tentative program of the ACOG, Dr. Robert W. Kistner, Assistant Clinical Professor of Obstetrics and Gynecology at Harvard University School of Medicine, will speak on "Induction of Labor with Clomiphene Citrate," "Clinical Application of Synthetic Progestins," "Hormonal Treatment of Endometriosis," and "Culdoscopy — An Integral Part of the Infertility Survey." Dr. Munsick's subjects will be "Use of Posterior Pituitary Extracts in Obstetrics and Gynecology," and "Prenatal Estimation of Fetal Weight."

Officers of the New Mexico Chapter of the AAGP are: Dr. James A. Koch, Albuquerque, President; Dr. Herschel L. Douglas, Lovington, Vice-President; Dr. Paul Feil, Deming, President-Elect; and Dr. C. W. Carroll, Las Cruces, Secretary-Treasurer.

Headquarters for the joint meeting will be the Chaparral Motel in Ruidoso.

American Fracture Ass'n to Meet in Venezuela

The 27th annual meeting of the American Fracture Association will meet this Fall in Caracas, Venezuela, at the Macuto-Sheraton Hotel, November 1-5, 1966, Dr. W. Compere Basom, El Paso, President, has announced.

Outstanding speakers on the program are Dr. Oswaldo P. Campos, Rio de Janeiro, Brazil; Dr. Jorge Garcia Arosemena, Panama City, Panama; Dr. Eduardo Alcivar, Guayaquil, Ecuador; Dr. Alfonso Montagne, Lima, Peru; Dr. Ramon I. Fernandez-Torres, Caracas; Dr. Jorge Figarella, Caracas; and Dr. Luis Irigoyen Dotti, Barquisimeto, Venezuela.

Members on the program from the United States include Dr. Dana Street, Professor of Orthopaedic Surgery, University of California at Los Angeles; Dr. Robert Mazet, Jr., Chief, Orthopaedic Surgery, Veterans Administration Hospital, Los Angeles; Dr. Russell D. Harris, Oklahoma City; Dr. Robert B. Elliott, Houston; Dr. Earl D. McBride, Oklahoma City; Dr. J. J. Toland, III, Philadelphia; Dr. Milton C. Cobey, Washington, D. C.; Dr. Garrett Pipkin, Kansas City, Missouri; and Dr. Roger Anderson, Seattle, Washington.

One entire day will be spent in attending a meeting to be held by the University of Caracas School of Medicine, under the sponsorship of the Orthopaedic Staff. A post-meeting tour and program has been arranged for San Juan, Puerto Rico, from November 6-10.

All physicians interested in Orthopaedics and in touring Venezuela and Puerto Rico, contact the Vincennes Travel Service, 405 Main Street, Vincennes, Indiana.

Vascular Surgery is Subject of Carlsbad Meet

"Recent Developments in Vascular Surgery" will be the subject at a one-day meeting of the Eddy County Medical Society, June 1, 1966, in Carlsbad, New Mexico. Dr. Jesse E. Thompson, Associate Professor of Surgery at Southwestern Medical School in Dallas, will be the visiting speaker. Presentation and discussion of cases will be held at the St. Francis Hospital Staff Room from 2-5 p.m. At a dinner meeting at 7:30 p.m. in the Riverside Country Club, Dr. Thompson will discuss the subject for the day's session. Dr. T. E. Hauser of Carlsbad is in charge of the scientific program.

Cardio-Respiratory Resuscitation By Mobile Intensive Care Unit

A new cardio-respiratory resuscitation vehicle which may enable hospital emergency teams to save many lives has been used over 60 times for treatment of cardio-respiratory arrest at Pennsylvania Hospital in Philadelphia. Nicknamed Max*, the vehicle is designed to serve smaller hospitals as a mobile intensive care unit.

A conventional emergency cart brought to the patient's bedside usually takes six to eight persons over three minutes to set up.

With the new unit, rather than surrounding the patient with cumbersome equipment, the patient is placed on the vehicle's litter surface to become part of a man-machine system. The relationship of equipment and emergency team to the patient is such that it takes three or four persons only 26 seconds to complete the same lifesaving tasks.

Designed to be activated electronically by a newly developed Hospital Emergency Command System which will provide a simultaneous and automatic mobilization of equipment, personnel and elevators, Max is rushed to the emergency site. An elapsed time meter, monitoring scope and data acquisition recorder begin to operate at the time of the alert. As the four team members arrive, the patient is quickly transferred to the electrically isolated litter surface. The respirator, pneumatically powered external cardiac compressor and EKG needle electrodes are applied simultaneously and the patient's heart trace is immediately visible on the already operating cardioscope. A surgical cutdown on a vein to provide a route for intravenous drugs and endotracheal intubation to assure an open airway immediately follow.

The defibrillator may be used to electrically shock a quivering (fibrillating) heart into a normal rhythm or a pacemaker may provide periodic impulses to stimulate a heart beat.

Drugs, surgical instruments and other equipment are stored in compartmentalized drawers and cabinets which, once opened, cannot be closed without a special control key. This provides an effective check and inventory system to insure that Max is always fully replenished with fresh drugs and instruments.

Self-contained oxygen and electric power permit the respirator, external cardiac compressor, cardioscope and other equipment to sustain the patient while he is moved to an intensive care unit for post resuscitation care or to an operating room for emergency surgery. During surgery Max continues to support and monitor the patient who remains on the unit which also serves as the operating table.

In the not-so-distant future, when transplantation techniques are in widespread use, Max may sustain and transport a dying patient to the operating room to receive an artificial heart.

Max's data acquisition system automatically records the emergency team's orders and activities and physiologic data (EKG, EEG, pulse pressure), from the patient. These records provide information for evaluation of techniques and research in resuscitation physiology and emergency care organization.

The system was designed by Joel J. Nobel, while serving as a resident in surgey at Pennsylvania Hospital.

— Reprinted from Philadelphia Medicine, February 20, 1966.

*The MAX cart was designed by Smith Kline & French Laboratories medical instrumentation subsidiary, Corbin-Farnsworth, Inc. of Palo Alto, California, in collaboration with Dr. Joel J. Nobel.

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IN THIS ISSUE

Ninth Annual

RUIDOSO SUMMER CLINIC

and

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Chaparral Motel — Ruidoso, N. M.

July 25-30, 1966

COMPLETE PROGRAM ON PAGE 166

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June, 1966

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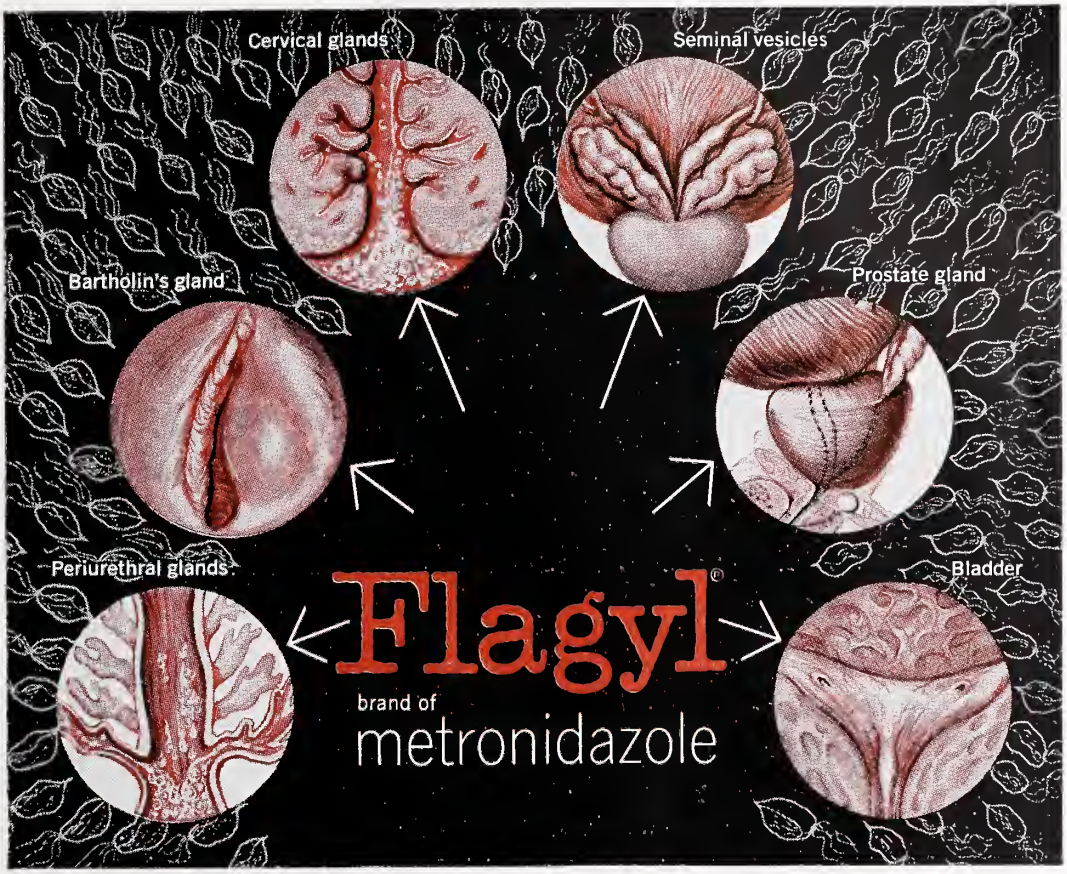

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Precautions and Side Effects—Complete blood cell counts should be made before and after therapy, especially if a second course is necessary.

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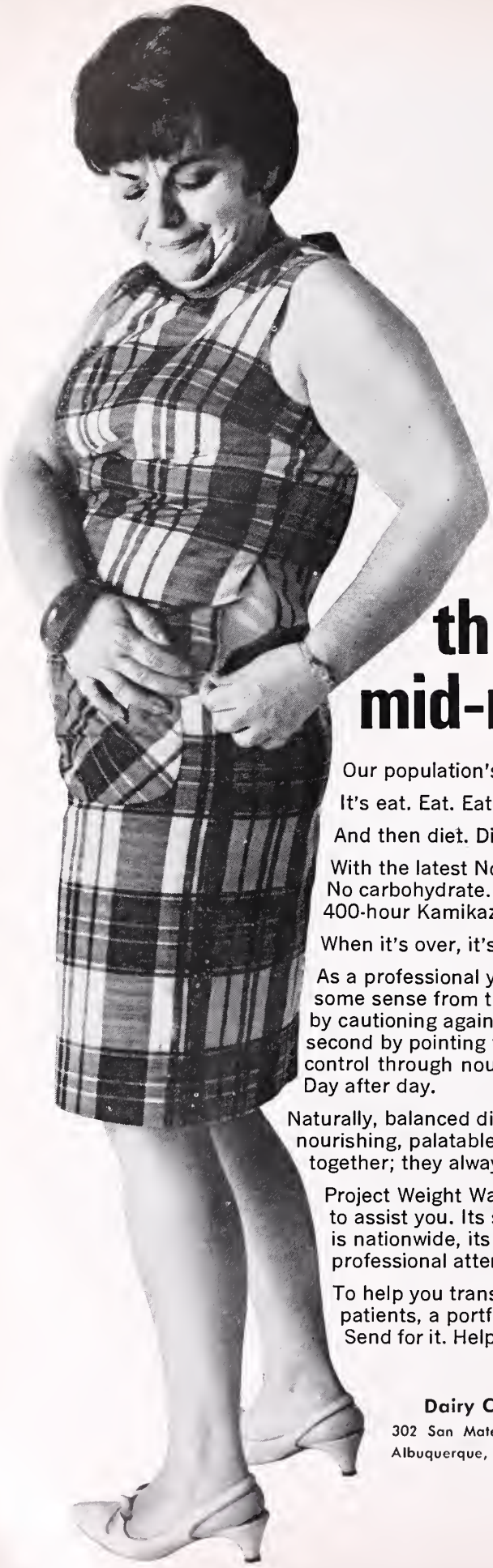

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PROJECT WEIGHT WATCH

FROM THE DOCTOR'S LOUNGE . . .

The Rape of the Tax Payer: RCC — Ratio of Cost to Charges

Sol Heinemann, M.D., *El Paso*

When the proponents of Medicare were selling the tax payer on the idea, one of their big talking points was that it would take the financial burden off the young family with children and by a process of equitable taxation spread the financial care of the elderly throughout the population of wage earners.

In the Wall Street Journal of May 2, 1966, Jonathan Spivak reports that the Social Security Administration will now unveil its arrangements for paying the hospitals under Part A of the Medicare law. According to this report, the Social Security Administration will use RCC, or a computation of costs based on the ratio of hospital charges to hospital costs. The Social Security Administration reasons that since its clients will not be using certain hospital facilities that are money losers, they should not pay any part of the cost of these departments. This includes maternity services, pediatric services, etc. In all communities the hospital is an enterprise for the care of illness, and medical problems of the entire community. Based upon this, private health insurance has always absorbed this cost in the daily average rate paid the hospital. Now we are told that these added costs of the departments that make a hospital a well rounded institution should be borne by those that use them. *This in truth is double taxation of the wage earner,* who will pay for Medicare through taxes, and then be forced to pay increased hospitalization, and increased health insurance rates for the care of his family. It looks like the voter has again been played for a sucker.

It is interesting to note the government does not keep books using RCC. The Veterans Administration does not differentiate between service connected and none-service connected illness. Military hospitals do not differentiate between military personnel and their families. In fact, no Utilization Committee or rules for utilization have been set up for government hospitals, as they do not come under the Medicare Law.

The Social Security Administration states that this new bookkeeping system will be put into effect within the next 18 months. It looks like hospitals will have two sets of charges. One charge for Medicare patients that will not include any department they do not use, and another charge for the remainder of the population who will be burdened with maintaining these departments in order that our hospitals shall be complete medical facilities.

While I understand the idea behind RCC is to save the tax payers' dollar and keep down the cost of Medicare, its results will be to raise the tax payers' hospital costs and health insurance rates. In the long run it will probably force him to vote for Medicare for all people to get relief from this increased burden of double taxation.

Possibly in order to save our hospitals from coming totally under federal control the idea should be promulgated that the government build and run its own hospitals for Medicare recipients. Based on present Veterans Administration costs Medicare would go broke in a hurry, but it would at least save the tax payer and keep his hospitals running at a more reasonable cost for him.

People under Medicare are no longer any different from missiles built by an aircraft corporation. They are a government contract and as such should pay their fair share. If the government cannot do this under the present Medicare rates that they are setting then it should raise the rates rather than put the burden on the community to pick up deficits of running a well rounded hospital. This is a fight that organized medicine should lead in defense of the public.

1900 N. Oregon

Ruidoso Clinic—Ob-Gyn Program Announced

Officials of the New Mexico Chapters of the American Academy of General Practice and the American College of Obstetrics and Gynecology have announced the complete program for their joint meeting at the Ninth Annual Ruidoso Summer Clinic in Ruidoso, New Mexico. Dates for the AAGP sessions are July 25 through 28, 1966; ACOG sessions are set for July 29 and 30.

Attending physicians will earn a total of 22 hours of Category I credit for their attendance at the six morning sessions of the joint meeting. The Faculty for the Ruidoso Clinic will be provided by the University of New Mexico School of Medicine. The meeting is sponsored by the New Mexico Chapter of the American Academy of General Practice.

The speakers for the Ruidoso Clinic from the University of New Mexico School of Medicine are Dr. Martin Brandfonbrener, Associate Professor of Medicine; Dr. Frederick Cohn, Adjunct Instructor of Obstetrics and Gynecology; Dr. Theodore N. Finley, Associate Professor of Medicine; Dr. Arnold H. Greenhouse, Associate Professor of Medicine; Dr. William R. Hardy, Assistant Professor of Medicine; Dr. Alexander L. Kisch, Assistant Professor of Medicine; Dr. Eugene L. Klingler, Jr., Instructor in Medicine; Dr. John K. Leach, Assistant Professor of Medicine; Dr. A. North Longfield, Associate Professor of Medicine; Dr. William S. Lovekin, Assistant Professor of Medicine; Dr. Robert A. Munsick, Professor of Obstetrics and Gynecology; Dr. Solomon Papper, Professor of Medicine; Dr. Robert S. Stone, Professor of Pathology; Dr. Carlos A. Vaamonde, Instructor in Medicine; and Dr. Robert Whang, Assistant Professor of Medicine. Panel moderators will be Dr. Reginald H. Fitz, Dean and Professor of Medicine and Dr. George M. Boyden, Assistant Dean of Medicine and Assistant Professor of Medicine at the School.

The special guest speaker at the ACOG Meeting is Dr. Robert W. Kistner, Assistant Clinical Professor of Obstetrics and Gynecology at Harvard University School of Medicine.

Officers of the New Mexico Chapter of the AAGP are: Dr. James A. Koch, Albuquerque, President; Dr. Herschel L. Douglas, Lovington, Vice-President; Dr. Paul Feil, Deming, President-Elect; and Dr. C. W. Carroll, Las Cruces, Secretary-Treasurer.

The registration fee for the Ruidoso AAGP meeting is $30. This does not apply to Residents, Internes, Medical Students, and Military and Public Health Service personnel. There will be no charge for the ACOG meeting.

Dr. George M. Boyden of the University of New Mexico School of Medicine is in charge of arrangements.

Headquarters for the joint meeting will be the Chaparral Motel in Ruidoso.

The complete programs are as follows:

Ruidoso Summer Clinic
Monday — July 25
Subject: Renal Disease

8:00 a.m. Registration
Lobby, Chaparral Motel

8:30 a.m. Welcome
James A. Koch, M.D., President
New Mexico Chapter AAGP

8:40 a.m. Evaluation of a Patient with Renal Calculi
Robert Whang, M.D.

9:10 a.m. Practical Aspects of the Diagnosis of Acute Renal Failure
Carlos Vaamonde, M.D.

9:40 a.m. Technique and Indications for Peri-

toneal Dialysis
Eugene L. Klingler, Jr., M.D.

10:10 a.m. Coffee

10:30 a.m. The Nephrotic Syndrome
Solomon Papper, M.D.

11:10 a.m. Panel: Renal Disease
Moderator: Reginald H. Fitz, M.D.
Panelists: Drs. Klingler, Papper, Vaamonde and Whang

Tuesday — July 26

Subject: Cardiovascular Disease

8:30 a.m. The Indications for Cardiac Catherization
John K. Leach, M.D.

9:00 a.m. Drugs and Hypertension
William S. Lovekin, M.D.

9:30 a.m. Diet and Other Factors Influencing Coronary Disease
Martin Brandfonbrener, M.D.

10:10 a.m. Coffee

10:30 a.m. Current Concepts in Cerebral Vascular Disease
Arnold H. Greenhouse, M.D.

11:10 a.m. Panel: Cardiovascular Disease
Moderator: Solomon Papper, M.D.
Panelists: Drs. Brandfonbrener, Greenhouse, Leach and Lovekin

Wednesday — July 27

Subject: Pulmonary Disease

8:30 a.m. Treatment of Chronic Lung Disease
A. North Longfield, M.D.

9:00 a.m. Antibiotics and Pneumonia
Alexander L. Kisch, M.D.

9:30 a.m. Viral Diseases of the Lung
Robert S. Stone, M.D.

10:10 a.m. Coffee

10:30 a.m. Treatment of Acute Respiratory Failure
Theodore N. Finley, M.D.

11:10 a.m. Panel: Pulmonary Disease
Moderator: Solomon Papper, M.D.
Panelists: Drs. Finley, Kisch, Longfield and Stone

Thursday — July 28

Subject: Obstetrics and Gynecology

8:30 a.m. The "Pill" as Related to Contraception and Menopause
Frederick Cohn, M.D.

9:00 a.m. Hypofibrinogenimea in Pregnancy
William R. Hardy, M.D.

9:30 a.m. Urinary Tract Infection in Pregnancy
Alexander L. Kisch, M.D.

10:10 a.m. Coffee

10:30 a.m. Normal and Abnormal Labor
Robert A. Munsick, Ph.D., M.D.

11:10 a.m. Panel: Obstetrics and Gynecology
Moderator: George M. Boyden, M.D.
Panelists: Drs. Cohn, Hardy, Kisch and Munsick

Ob-Gyn Seminar

Friday — July 29

Presiding: Howard L. Smith, M.D., Albuquerque, Regional Governor, ACOG

9:00 a.m. Hormonal Treatment of Endometriosis
Robert W. Kistner, M.D.

9:40 a.m. Endocrine Evaluation by Means of Vaginal Cytology
Duane W. McCarty, M.D., Albuquerque

10:20 a.m. Coffee

10:40 a.m. Surgical Treatment of Endometriosis
Robert W. Kistner, M.D.

11:20 a.m. Panel Moderator:
Benjamin N. Branch, M.D., Albuquerque

Saturday — July 30

Presiding: James A. Koch, M.D., Albuquerque, President, New Mexico Chapter AAGP

9:00 a.m. Clinical Application of Synthetic Progestins
Robert W. Kistner, M.D.

9:40 a.m. Disorders of Sexual Differentiation
Michael J. Perley, M.D., Albuquerque

10:20 a.m. Coffee

10:40 a.m. Induction of Ovulation — Observations on the Use of Clomid, Pergonal and Chorionic Gonadotropin
Robert W. Kistner, M.D.

11:20 a.m. Panel Moderator:
Benjamin N. Branch, M.D.

Growth and Death in a Small Hospital

C. Herbert Fredell, M.D.,* *Flagstaff, Arizona*

Small hospitals play an increasingly important role in the rendering of good medical care. Improved physical plant and facilities, together with an increase in the number of trained personnel, have made the improved quality of patient care a reality. Most small hospitals have problems that differ from those of larger institutions. Basically, they are geographically located in smaller clusters of population; are financed in a different manner; are dependent on a smaller labor pool, and care for several problems more infrequently than their larger counterpart.

The professional staff is often smaller in number and more limited in its scope of training. The urgent problems that must be cared for by the staff and hospital are often similar to those in the large hospital. However, problems that are not emergencies in nature will vary somewhat from those cared for in larger medical facilities.

Nine Year Study

Because of the difference in environment one finds in a small hospital setting, and because of the need to evaluate the results of care in this setting, this study was initiated. It covers a period of nine years.

The background of the study has been one of growth in the Flagstaff community and its hospital. The community grew from 17,000 to 24,000 in population during this period. It is situated in the north central portion of the state and has a single small hospital, the Flagstaff Hospital. The hospital has grown more in modernization of facilities than in bed capacity during this time. A new Obstetrical Unit and a new Surgical operating-room suite have been added. Obsolete beds were replaced by modern accommodations for patients. At present the bed capacity is 53 adult beds and 20 bassinets.

Within the institution, basic medical specialties are represented by trained staff members who render a wide variety of care. Because of the hospital's size and location, it must function as a general hospital, largely for short-term care. The average patient stay is 4.2 days. Confronted with increasing need for service, the hospital is currently in the midst of a major expansion.

The following tabulation of information concerning causes of death, frequency of autopsy, age, sex, and relative frequency of certain diseases has not been presented to prove any particular thesis.

A Profile

This is a profile of a small hospital in a period of growth and transition. During the time of this study, the hospital underwent two expansions in bed capacity and specialized facilities, the medical staff doubled in number, and basic specialties became represented. The impact on the community's medical care has been favorable.

Before embarking on a new phase of hospital

*Chief of Surgery, Flagstaff Hospital, Flagstaff, Arizona.

care within a larger and more modern environment, it seems appropriate to look back over the steady growth as measured by the number of hospital discharges, thus indicating the number of people served, and simultaneously to consider one of the measures of the results of care rendered to these people.

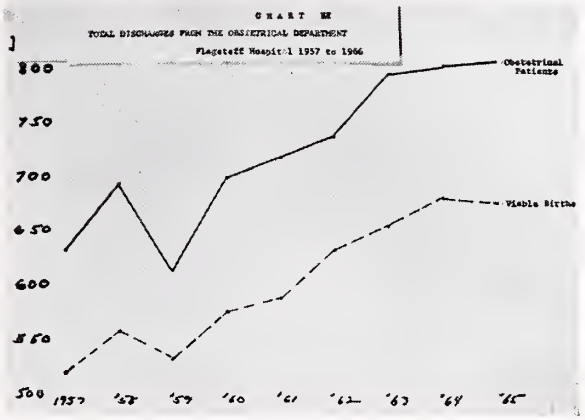
The most unsatisfactory result a physician can have in the care of a sick patient is the demise of that patient. Often the physician takes a look in hindsight to see if he might learn something that might be helpful in the future care of similar problems. An awareness of the causes of death in a particular hospital environment is often of value. The information here has been totaled on an annual basis for comparison over the nine-year-period.

Admittedly a single measurement, such as the death rate, is not a fair evaluation of the quality of patient care or the adequacy of professional care and judgment. It is, however, a harsh yardstick that people apply to an institution and its related personnel in measuring results. Other measurements are well known to medical personnel and laity alike.

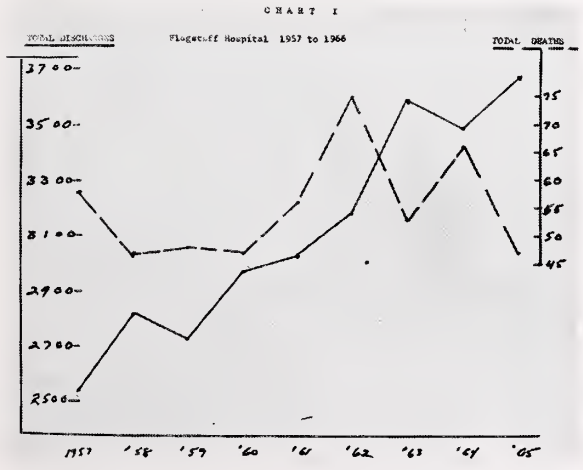
Within the scope of this study we find the number of hospital discharges on a steady increase (Chart I). The number of deaths annually was at a peak during 1962, but since then it has shown a progressive decline. This has occurred in spite of the increasing scope of the hospital's activity and the increasingly wider variety of medical problems being treated. With a wider variety of serious and more dangerous illnesses being cared for, one might expect a higher mortality rate.

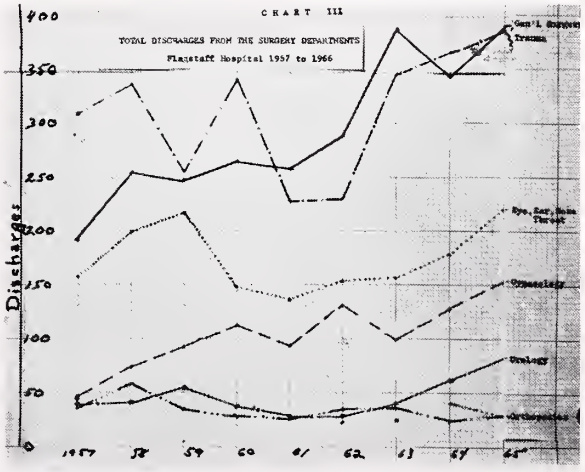
No Maternal Deaths

During the period of this study the Obstetrical Department has had no maternal deaths. A steady increase in the number of Obstetrical patients discharged from the hospital has occurred (Chart II). The number of viable births has shown a steady increase until last year when it decreased slightly.

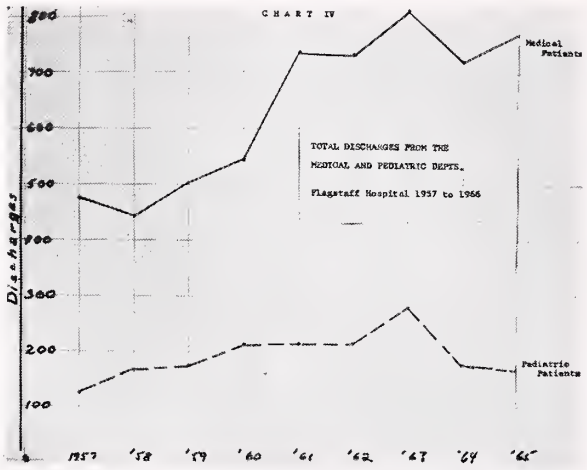


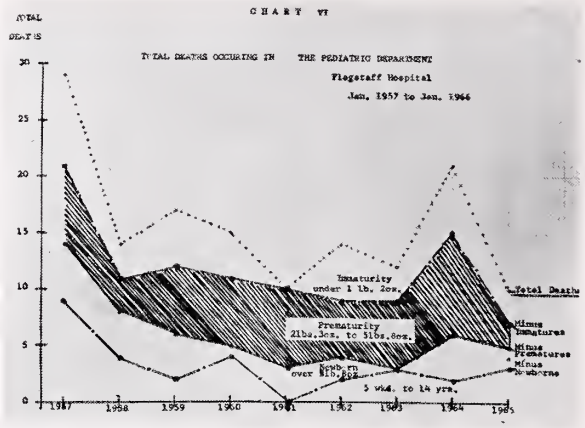
Patients discharged from the Surgery Department have shown an increase in number in all categories except Orthopaedics (Chart III). A significant number of patients with problems related to trauma have been discharged. In such a community as Flagstaff, the increasingly important role played by the small hospital with cases of acute trauma is apparent.





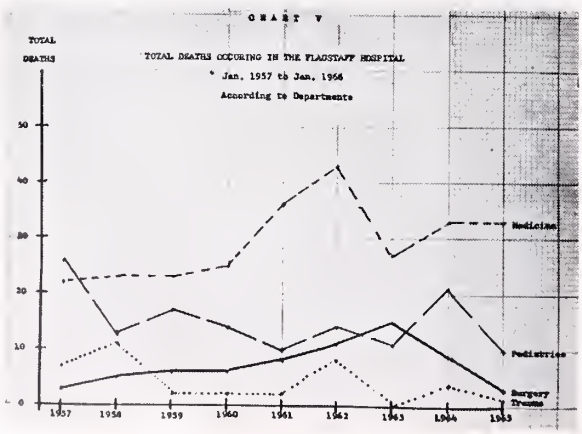
Within the Flagstaff Hospital there is no department devoted totally to the care of Pediatric patients. Casting the Pediatric patient into the general hospital milieu has been reflected in the lack of growth in hospital discharges of Pediatric patients. A hospital located in an expanding community would logically reflect this growth in its Pediatric Department. In Flagstaff this did not occur (Chart IV).

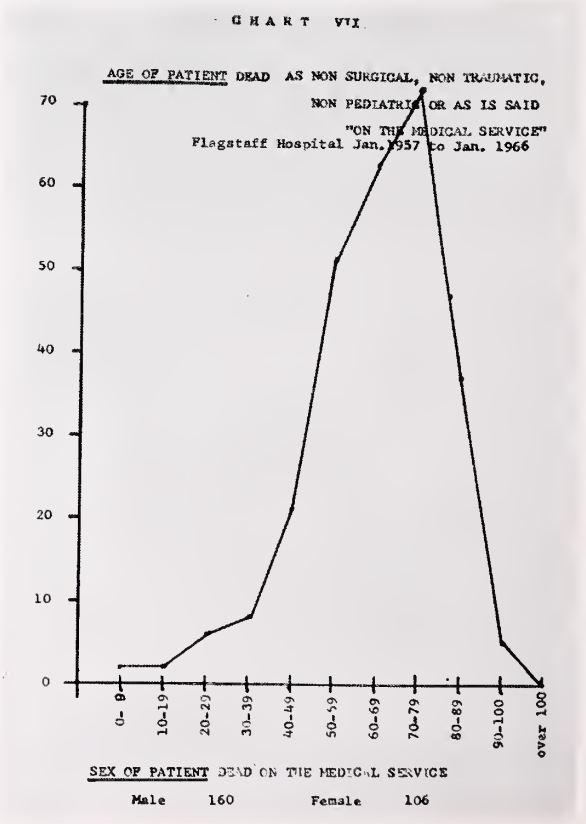


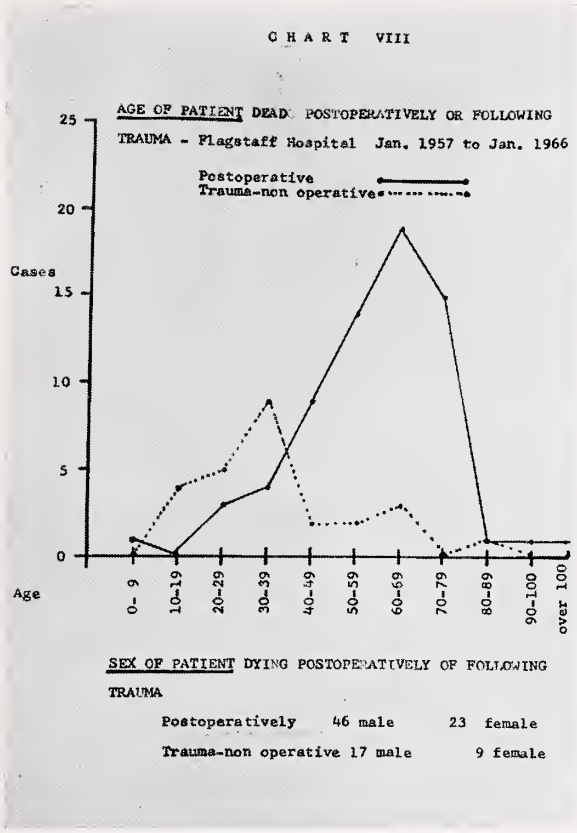


In the last five years the Internal Medicine Department has discharged almost the same number of patients every year. Growth within this area of care also has not been as progressive as might be expected. However, the overall trend for nine years is upward.

In addition to the study of the number of patients discharged from the various services, a compilation of the number of deaths occurring annually within each department was done (Chart V). It can be seen that there have been certain years that particular departments had a larger than usual number of patient deaths. The decline in the number of deaths occurring postoperatively, in spite of a steady increase in the number of patients operated on, has been encouraging. The increase in deaths during 1964 on the Pediatric service can be pinpointed to a larger number of premature infant deaths that year (Chart VI).







The cause of death has been tabulated according to the three basic departments within the hospital (Tables I, II and III). One may note that death occurred in the Pediatric age group of patients because of bronchopneumonia and gastroenteritis. Infection remains a significant cause of death in children.

Table I

Causes of Death Occuring in the Pediatric Department of the Flagstaff Hospital
Jan. 1957 to Jan. 1966

	142 Deaths (56 Autopsies) Pediatrics	Newborn	Premature
Hyalin Membrane Disease		8 (6)	11 (10)
Congenital Atelectasis		4 (3)	5 (4)
Pneumothorax (Lung Cysts 2)		6 (5)	
Pneumonitis (Aspiration)		4 (3)	2 (2)
Bronchopneumonia	15 (6)		
Congenital Heart Disease		3 (3)	2 (2)
Multiple Congenital Abnormalities		1 (1)	
Anencephalus			1
Hydrocephalus	1		
Congenital Diaphragmatic Hernia		1 (1)	
Erythroblastosis Fetalis		2 (1)	1
Coeliac Disease	1 (1)		
Prematurity			22 (3)
Immaturity			30
Gastroenteritis-dehydration-septicemia	7 (2)		
Bacteremia-Septicemia		1 (1)	1
Brain Abcess	1 (1)		
Brain Injury & Subdural Hematoma	1 (1)		
Salicylate poisoning	2		
Hemorrhage from untied umbilical cord			1
Leukemia	1		
Malnutrition	1		

Table II

Causes of Death Occuring in the Medical Department of the Flagstaff Hospital
Jan. 1957 to Jan. 1966

	265 Deaths	95 Autopsies
Acute Myocardial Infarction	75	(30)
Congestive Heart Failure	27	(6)
Arteriosclerotic Heart Disease (heart block 4)	14	(3)
Cerebral Vascular Accident (acute subarach. 3)	46	(13)
Dissecting Aortic Aneurysm	1	(1)
Mesenteric Thrombosis	2	(2)
Carcinomatosis	54	(12)
Unspecified 10, Prostrate 3, Breast 7, Lung 5, Stomach 3, Ovary 2, Brain 3, Cervix 2, Pancreas 2, Liver 2, Vulva 1, Gall Bladder 1, Small Bowel 1, Colon 1, Uterus 1, Esophagus 1, Kidney 2, Cordoma 1, Melanoma 1, Leukemia 2, Lymphosarcoma 1, Hodgkins 1, Multiple Myeloma 1.		
Renal Failure—Arteriosclerotic renal disease	6	(3)
Chronic glomerulo or pyeloneph.	4	(3)
Liver Failure—Cirrhosis—(esoph. varices-hem. 3)	7	(3)
Bronchopneumonia	15	(9)
Emphysema	3	(3)
Pulmonary Infarction	2	(2)
Diabetic coma	1	
Insulin shock	1	
Overdoseage Ingestion (Aspirin 1, Baking soda 1)	3	(1)
Periarteritis Nodosa	2	(1)
Lupus Erythematosis	1	
Hemachromatosis	1	(1)
Von Recklinghausens Disease (Adrenal hem.)	1	(1)
Gastric Hemorrhage Site ?	1	(1)

Table III

Causes of Death Occuring in the Surgery Department of the Flagstaff Hospital
Jan. 1957 to Jan. 1966

	94 Deaths	51 Autopsies
Trauma		
Severe Brain Damage	19	(4)
Crushed Chest—Multiple Injuries	5	(4)
Abdominal Hemorrhage—Multiple Injuries	2	(2)
Burns	1	
Postoperative		
Brain Damage Due to Trauma	5	(2)
Cerebral Vascular Accident	5	
Congestive Heart Failure	5	(3)
Cardiac Arrest	5	(4)
Myocardial Infarct	3	(1)
Cardiac Tamponade	1	(1)
Endocarditis	1	(1)
Bronchopneumonia	3	(3)
Empyema	1	
Pulmonary Infarct	1	(1)
Hypocarbia With Severe Emphysema	1	(1)
Liver Decompensation & Failure	6	(4)
Hemorrhage	4	(1)
Pancreatitis	5	(4)
Carcinomatosis	3	(1)
Renal Artery Thrombosis	1	(1)
Aortic Thrombosis	1	(1)
Peritonitis & Septicemia	16	(10)

Newborn Infant

The newborn infant had lung diseases that caused death in a large proportion of the total deaths. Prematurity and immaturity were responsible for a large number of infant deaths.

In the Department of Internal Medicine the leading causes of death throughout the nation are reflected, and acute myocardial infarction, cerebral vascular accident, and carcinomatosis lead the list. Significant in this series of cases is the number of deaths due to primary bronchopneumonia in spite of all available therapy (Table II).

Deaths which occurred following severe trauma and after surgery have been tabulated (Table III).

Head injury leads the list of the causes of death under trauma. Peritonitis and septicemia are leading causes of postoperative deaths. Diseases of the cardiovascular system lead the other causes of death following surgery.

If the postoperative deaths are analyzed on the basis of the type of surgery performed and the number of deaths occurring following a particular operative procedure, we find that certain procedures are followed by a significant mortality rate (Tables IV and V). It is noteworthy that there have been no deaths following hernia surgery, appendiceal surgery, bone and joint surgery, tonsillar surgery, and neck surgery. During the entire period of this study, two deaths were considered to be anesthetic deaths.

Table IV
Causes of Death Following Surgery According to Organ or System
Jan. 1957 to Jan. 1966

Deaths		Procedures
5	**Neurological**	
	Cranial trephines or elevation of compound depressed skull fracture	20
	Severe brain damage	4
	Cordotomy, Spinal cord decompression	4
	Bronchopneumonia	1
7	**Vascular**	
	Aneurysmorrhaphy, Aortic resection, Aorta suture	7
	Cardiac arrest	2
	Hemorrhage, dehiscence suture	1
	Hemorrhage massive transfusion	1
	Renal artery thrombosis/uremia	1
	Femoral artery prosthesis	11
	Cerebral vascular accident	1
	Mesenteric, iliac endarterectomy, renal artery surgery	9
	Congestive heart failure	1
2	**Thorax**	
	Thoracotomy	24
	Cardiac arrest/obstr'n bronchus	1
	Cardiac arrythmia/contusion	1
6	**Urogenital**	
	Prostate, kidney, bladder, ureter, uterus, tubes, ovaries, cesarean sections	646
	Myocardial infarction (prostate)	1
	Pulmonary emboli (prostate)	1
	Congestive ht. failure (prostate)	1
	Endocarditis (nephrectomy)	1
	Emphysema, hypocarbia (nephrolith)	1
	Bronchopneumonia (cystoplasty)	1
12	**Biliary tract and Liver**	
	Cholecystectomy, cholecystostomy, choledochotomy, biopsy	379
	Liver failure	5
	Pancreatitis	3
	Peritonitis	2
	Myocardial infarct	1
	Bronchopneumonia	1

Table V
Causes of Death Following Surgery According to Organ or System
Jan. 1957 to Jan. 1966

Deaths		Procedures
17	**Stomach**	
	Gastrectomy, Vagotomy & Pyloroplasty, Gastroenterostomy	236
	Congestive heart failure	3
	Peritonitis or bacteremia	4
	Pancreatitis	2
	Empyema	1
	Hemorrhage, massive transfusions	1
	Myocardial infarction	1
	Cardiac tamponade	1
	Liver failure	1
	Cardiac arrest	1
	Cerebral Vascular accident	1
	Aortic thrombosis	1
9	**Small Bowel**	
	Bowel resection, suture, enterolysis	90
	Peritonitis or septicemia	8
	Cerebral vascular accident	1
2	**Colon**	
	Colectomy, colostomy, suture of colon, cecostomy	71
	Peritonitis	1
	Septicemia after abscess drainage	1
2	**Spleen, Pancreas, Omentum**	
	Splenectomy, pancreatectomy, suture, excision	39
	Cerebral vascular accident	1
	Mesenteric thrombosis	1
4	Exploratory laparotomy alone, with biopsy, or incidental appendix	49
	Carcinomatosis	3
	Subarachnoid hemorrhage, crushed ribs	1

Comment

During a nine-year period a small hospital has grown along with the growth of the community. Certain areas of care within the hospital have not grown with the same rapidity as others. Problems unique to a small hospital influence the variability of growth and contribute to the profile of care reflected by that hospital. The Flagstaff Hospital has been no exception to the problems afflicting most small hospitals across the nation.

A study of the type reported in this paper serves several functions. It demonstrates to the administration of the hospital and the medical staff the growth rate and lack of growth in specific areas of patient care. It demonstrates the areas of patient care that are associated with specific death rates and the specific causes of death in these areas. Within the scope of this study no particular thesis was propounded. It was compiled to present a profile of the past to better enable the administration and hospital staff to plan more wisely for future growth and care.

When a small hospital is in transition and the community is growing in size, many problems arise in the course of progress. It is hoped that this study will better enable those involved in similar circumstances to plan for and cope with unpredictable circumstances as they arise.

This experience is not considered to be unique, yet the lack of documented experiences of this type causes one to wonder what the norms and expected norms in similar circumstances might be. Hopefully this study will stimulate others to report their experiences so that a wider sharing of opinions and recommendations may result.

The causes of death reflect the conditions most often associated with demise. The breakdown into different medical departments serves as a guide to physicians who work within those areas. Improvement in the results of therapy is constantly being sought by physicians, and in that respect certain diseases require increased efforts, particularly in the small hospital.

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CONTENTS

An Approach to Management of Status Epilepticus

Maj. Darrell S. Buchanan, MC,* *El Paso*

When two or more epileptic seizures occur without an intervening lucid interval, the patient is stated to be in Status Epilepticus. Occasionally, rather than suffering an apparent series of seizures linked by unconsciousness, the patient in status will seem to be suffering from a single, prolonged convulsion. In either case a medical emergency exists; prompt, effective treatment is mandatory to protect the patient from complications such as exhaustion, brain damage, and even death.

There are many drugs which can be used to terminate this state of "discontinuous epilepsy", and a number of vastly divergent approaches have been successful. However, it is all too frequent that status epilepticus is managed ineffectively during the critical first hour of the attack.

Status epilepticus is comparatively rare; frequently the physician faced with the initial management of this terrifying condition is not familiar with the details of the multiple methods of management which have been advocated. It is the purpose of this paper to propose a single "cook book" approach which has been found to be most effective for initial management.

This presentation will be concerned with grand mal status. Petit mal, Jacksonian motor and psychomotor status attacks do occur, but the urgency of treatment is not as pressing as in grand mal status. However, the method of therapy to be proposed has been found to be quite effective in these forms of status epilepticus also.

Diagnosis

Usually the patient presents no problem in diagnosis when arriving at the initial treatment area. He will usually be in coma or experiencing a generalized seizure. Occasionally patients will become stuporous and even combative between convulsions, but as long as seizures are joined by periods of altered consciousness, the diagnosis of status epilepticus is justified. Occasionally, if a patient has been in status for an hour or more, he may fail to exhibit the characteristic motor manifestations; the observer may not realize that the patient is in the throes of seizure activity. Careful continuing observation will usually reveal the paroxysms, but rarely the diagnosis of status can be established only by EEG examination. An extremely important consideration at the time of initial confrontation is vigorous investigation and proper evaluation of physical signs that could reveal underlying conditions which could provide clues indicating the etiology of status epilepticus. It is obvious that even initial management should be directed toward the basic etiology if status is due to hypoglycemia or anoxia (as produced by an aspirated foreign body). If status were precipitated by cerebral swelling as would occur in lead encephalopathy or acute encephalitis, it might be more reasonable to attempt to control the edema with such drugs as Mannitol, urea or corticosteroids. There are of course other rare instances where a preordained treatment program could be hazardous; the individual with acute intermittent porphyria might be placed in jeopardy by administering barbiturates. For the most part, status epilepticus is the immediate emergency which must be controlled; the question of etiology must wait.

What about the use of IV sodium amytal in the

*Chief, Neurology Service, William Beaumont General Hospital, *El Paso, Texas.*

treatment of status epilepsy? This drug is familiar to most physicians; it is used more frequently than any other agent in the management of status. It remains popular because in most cases it will control the seizures. However, in the occasional case where seizure control is not achieved, it complicates subsequent management because of its significant effect in depressing respiration. For this reason I strongly advocate that amobarbital* be abandoned as a treatment of status epilepsy.

Act Quickly

When the patient is first seen, the physician must act quickly. Evaluation of airway patency and respiratory adequacy is of utmost urgency. If no reliable historian is available the physician must seek bracelets, necklaces or tattoos to check if they carry pertinent medical information. An aide should search the patient's billfold or purse for medical information cards. As soon as it is apparent that the patient is in status epilepticus, 100 mg of phenobarbital (or 2-3 mg/lb if dealing with a child**) should be given IM. An IV portal should be immediately established; blood should be drawn for basic laboratory studies including a blood sugar determination. An infusion of D5W is started followed by slow administration of another 100 mg (or 2-3 mg/lb in a child) of phenobarbital. The attending physician should **never** leave the bedside of a patient in status until he is satisfied that the condition is controlled. Respiration may need mechanical assistance; the airway must be maintained. Perhaps most important, the sensing of the urgent requirement to control the seizures is much more easily achieved if one is standing beside the patient.

While waiting for 15 minutes to observe the effect of phenobarbital, the physicians and nurse should prepare for the next step of treatment. Intravenous paraldehyde has been found to be effective in almost all cases of refractory status epilepticus. A solution is prepared by mixing 10 cc of paraldehyde with 200 ml of D5W, normal saline or isolyte. The paraldehyde must be fresh and must come in sealed ampules. Bottled paraldehyde tends to deteriorate with age and exposure to air forming dangerous breakdown products. It is mandatory that glass syringes be used in handling pure paraldehyde since it will act as a solvent for certain plastics. Initially the paraldehyde may seem reluctant to go into solution with the diluent, but with a little shaking, this will be accomplished within a few minutes.

If the patient continues to have seizures 15 minutes after the IV phenobarbital, the paraldehyde solution should be started. The first 50 ml of solution should be run in rapidly. This should take about 5-10 minutes. Seizures usually are controlled at this point. Even in cases where the patient is on the verge of respiratory depression due to previous heavy medication with barbiturates, no additional respiratory depression is seen. If seizures are not controlled, the rapid administration should continue until respiratory depression develops or control is attained. Experience reveals that no more than 150 ml of the solution (7½ ml of paraldehyde) are required to arrest seizure activity. The whole procedure should require no longer than one hour. In those rare instances where seizures are not controlled within an hour, or if significant respiratory depression should develop, an experienced anesthetist or anesthesiologist should be called upon to administer general anesthesia.

Frequent Error

A frequent error in management is failure to continue adequate medication once the initial episode is controlled. If the patient has been under treatment for epilepsy the drugs and dosages he has been receiving must be determined, and these drugs must be re-instituted. A frequent cause of status is sudden discontinuation of anticonvulsant medications in a known epileptic. If the patient has not been receiving treatment with anticonvulsants, the following program of management has been found to be of value.

Once the status attack is controlled by rapid administration of paraldehyde, the speed of administration is decreased to a point where 200 ml of solution is given over an 8-12 hour period. This is actually a period of titration; the speed of administration may be increased if the patient becomes agitated; it should be slowed if the patient becomes less responsive or comatose. Intra-muscular phenobarbital should be continued at 100 mg every six hours (2-3 mg/lb q 6 h in a child).

Diphenylhydantoin* should be started as early

*Amytal.

**To give reliable guide lines for phenobarbital dosages in children is extremely difficult. In a child who is of low body weight for age or who has been treated with long term barbiturates, the 2-3 mg/lb may be an inadequate dosage.

*Dilantin.

as practical. It may be given intra-muscularly or rectally. If diphenylhydantoin capsules are to be employed rectally, they should be perforated several times with a pin before insertion. The initial IM or rectal dose of diphenylhydantoin should be 200 mq every 6-8 hours in adults. This route of administration should be continued until the patient is able to take oral medication. It is advisable to overlap full oral dosage with full IM or rectal dosage for 24 hours due to the slow rate of utilization of oral diphenylhydantoin; the dosage by mouth should be 300-500 mg per day. Diphenylhydantoin dosage in children is more of a problem due to the sensitivity of children under six years of age to this drug. The routine use of diphenylhydantoin is not recommended in this age group. In children over six, 2-3 mg per pound is a reasonable dosage. After the patient has been seizure free for 24 hours, paraldehyde can be gradually discontinued.

"Cook Book" Approach

It is quite true that a "cook book" approach will not be suitable in every case. Certainly if an IV portal cannot be established one should immediately proceed to IM or rectal routes. Paraldehyde is quite effective by either of these routes. (If given rectally, paraldehyde should be mixed in equal volume with olive or corn oil. Dosage rectally is .3 ml of paraldehyde per pound of body weight not to exceed 15 ml; IM dose is .15 ml/lb not to exceed 10 ml.)

The basic principles of any approach in the management of status are: (1) rapid control of the seizures; (2) maintenance of a steady blood level of anticonvulsant medication. The most serious error is to employ intermittent high dosages of short acting barbiturates resulting in the patient alternating from barbiturate-induced coma with respiratory depression to repeated episodes of status.

Finally, the remarkable safety of paraldehyde used intravenously should be mentioned. There are case reports in the older literature of death due to IV, oral, IM or rectal paraldehyde. Several were related to the use of intravenous paraldehyde; the basic pathologic change causing death was acute pulmonary edema with pulmonary hemorrhage. In most instances rather large dosages of paraldehyde were given within short periods of time. Pulmonary or cardiac complications have not been seen in patients receiving paraldehyde given as suggested in this paper. Even evidence of pulmonary irritation as manifested by cough has not occurred. Serious pulmonary complications are most likely caused by rapid administration of undiluted or less diluted paraldehyde in doses far greater than have been suggested. There are reports of the sclerosing effect of paraldehyde on veins. Again this has not been observed with diluted paraldehyde. Avoidance of paraldehyde which has oxidized to other compounds has already been mentioned.

Hepatic insufficiency is considered a relative contraindication to the use of paraldehyde. Since about 80 per cent of paraldehyde is detoxified by the liver in normal man, a given dose would be expected to produce a more profound and prolonged effect in the presence of liver disease. Bronchopulmonary disease may also be considered a relative contraindication.

In summary a feasible program for the management of status epilepticus has been outlined. There can be no substitute for a high index of clinical suspicion, rapid institution of treatment, diligent personal management and continuous attention to details of therapy in the period following arrest of status. The use of phenobarbital and paraldehyde as the cornerstones of drug therapy has been recommended.

References

1. Lennox, W. G.: Epilepsy and Related Disorders, Little, Brown and Company, 1960.
2. Burstein, C. L.: The Hazard of Paraldehyde Administration, JAMA, Vol. 121 #3 p 187, (Jan. 16) 1943.
3. Goodman, L., and Gilman, A.: The Pharmacological Basis of Therapeutics, MacMillan Co., New York, P 178-180, 1941.

Peripheral Vasodilatation in Response to Controlled-Release Nitroglycerin

Harold Lepow, M.D., F.C.P.C.,* and Robert A. Turner, Ph. D.**

Introduction

For almost a century, nitroglycerin has remained the only undisputed antianginal agent and has served as a standard of comparison for all other drugs advocated for the treatment of the anginal syndrome.[1]

It has long been assumed that the antianginal activity of the esters of nitric acid is the direct result of coronary vasodilatation, allowing a rise in the myocardial blood flow. That the caliber of the coronary vessels can be increased by some organic nitrates has been clearly demonstrated in angiographic studies.[2,3] More recent evidence strongly supports the earlier concept that nitroglycerin acts as a potent, benign, coronary vasodilator.[5]

When nitroglycerin was therapeutically successful, some favorable effect on the heart's circulatory dynamics was believed to occur.[7] Whether the benefit is consequent to a simple vasodilatation of a portion of the coronary arterial system or to a more complex mechanism is not completely known.

The fact remains that for more than 80 years, no drug has proved superior to nitroglycerin in the treatment of angina pectoris.[7]

The frustrations in establishing the physiological events related to the treatment of angina pectoris have caused investigators of the disease to resort to difficult procedures involving visual angiography,[3] and the measurement of the time required for radioactive gases to pass through the coronary vessels. The duration of those experiments must be brief. Their performance demands great skill, but with good fortune they yield interesting results.

In studying long-acting vasodilators, the new procedures are not appropriate, and we have turned to other techniques. This report describes our experiments with nitroglycerin in controlled-release form*, which were designed to measure vasodilatation at the periphery. The usefulness of the controlled-release tablet in the prophylaxis of angina pectoris has been reported in clinical studies.[6,4]

Method

Several male and female subjects were used in this study of dermal temperature. It was recognized early in the investigation that few persons among the many tested were suitable for measuring dermal changes in temperature because they showed little response or an irregular response; that is, their homeostatic mechanisms were strong. The vasomotorial responses are influenced by many nervous and hormonal mechanisms, and vasomotion tends to be brief, with a return to the state existing before the addition of a pharmacological agent. The subjects whose temperature changes are reported here may be termed vasospastic. Some had a diagnosis of coronary infarction; others complained of cold fingers after slight ex-

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*Nitrong tablets were supplied by Wharton Laboratories, Inc., Division of U.S. Ethicals, Inc., Long Island City, New York.

posure to a cold environment.

The persons used in this study were maintained at rest in a reclining position, lightly clothed, and were instructed to consume only a light breakfast before the test. During the test period of 12 hours, they ate and drank little. They moved about very little. Ambient conditions were maintained at 72F and at 40-60 per cent relative humidity.

A dermal probe was affixed to a site on the skin with a wire connected to a Tele-thermometer.* After a period of 30-60 minutes of temperature recordings, in order to obtain the resting temperature, the subject was given two controlled-release tablets which indicate at zero hour.

This study was greatly aided by C. G. Herdenstam, M.D., Department of Cardiology, Sodersjukhuset Hospital, Stockholm, Sweden, who directed our attention to the use of dermal temperature changes as a measure of vasomotorial response. Some of the subjects were in the care of Dr. Herdenstam.

The probes were attached with adhesive tape to various sites of the body — the five fingers, the axilla, the great toe, and the medial aspect of the upper arm — and sometimes to several sites simultaneously. In general, the fingertip was the most satisfactory. Figure 1 shows the change in digital temperature over a period of 12 hours.

Nitroglycerin (controlled-release or sublingual) was taken at the points indicated.

The effect of sublingual nitroglycerin (1/150 grain, 0.43 mg) on dermal temperature was also measured. The results indicated that a temperature rise may be observed within 7-14 minutes. Thereafter the temperature returned quickly to the ante-ingestion level. Figure 2 shows the type of temperature curve obtained following the administration of sublingual nitroglycerin.

In order to test the response of nitroglycerin several hours after taking controlled-release tablets, some subjects were given a sublingual tablet after 12 hours. The responses are shown in Figure 1.

Results

The results indicate that in the case of vasospastic subjects a temperature rise occurs about 30 minutes or more after the administration of the controlled-release tablet and that the temperature remains elevated for about 12 hours before falling toward the ante-ingestion level. Thus, the temperature readings indicate that after administration of the tablets of controlled-release nitroglycerin, peripheral vasodilation persists for about 12 hours.

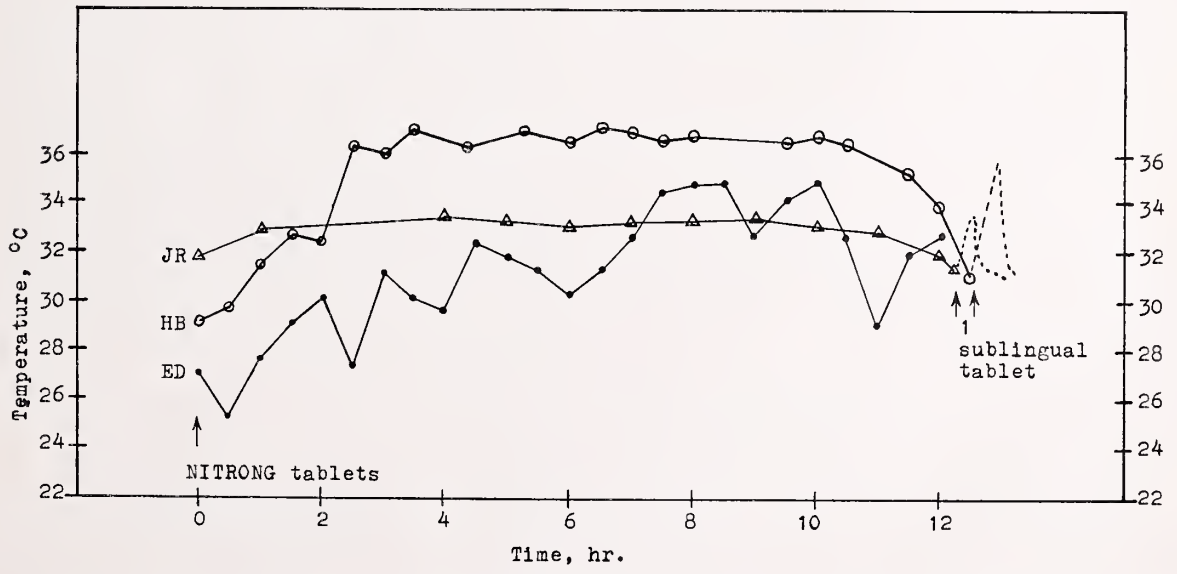

Fig. 1. Vasodilatation Measured by Dermal Temperature, After Administration of Nitroglycerin.

*Model 47, manufactured by the Yellow Springs Instrument Co., Yellow Springs, Ohio.

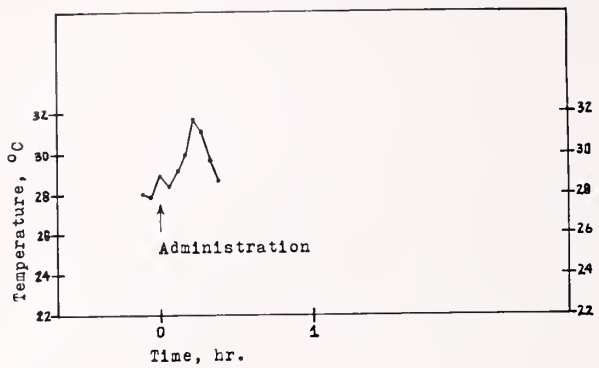

Fig. 2. Vasodilatation Measured by Dermal Temperature, After Administration of a Sublingual Tablet of Nitroglycerin.

It is interesting to note that the dermal temperature-rise, exemplified in Figure 1 correlates well with the blood nitrate-level for a 12-hour period.[9] Thus by two separate objective methods, the determination of blood nitrate-levels and the measurement of peripheral vasodilatation, the action of nitroglycerin for a period of about 12 hours is indicated.

The methods cited above for the clinical evaluation of anti-anginal drugs are valuable for different purposes and successful under the direction of some investigators. Searching for an objective measurement of vasodilatation, we have studied skin temperature. It is well known that blood flow increases with peripheral vasodilatation, causing a rise in dermal temperature; therefore, if a probe is attached to the skin, a rise in temperature may be taken as a measure of dilatation, provided that other variables are held constant.

The present study indicates a vasodilative response to nitroglycerin, in sublingual or controlled-release tablets, in susceptible, or vasospastic subjects.

Vasodilatation, as measured by increased skin temperature, lasted for 12 hours.

The results presented here are from subjects that showed changes in dermal temperature after the ingestion of nitroglycerin. However, the patients that did not show peripheral changes experienced the throbbing in the head, feeling of warmth, or headache known to follow the intake of nitroglycerin. There were qualitative responses to vasodilatation in some body parts (head, chest, and possibly viscera) of those subjects, even though vasodilatation in other parts (notably the skin) was not significant.

Summary

Nitroglycerin in a controlled-release tablet was administered to subjects whose dermal temperature was recorded during several hours by means of a probe attached to the skin. The temperature of susceptible subjects (that is, subjects having peripheral-vascular systems that respond readily without opposition by compensatory mechanisms) rose from the base-level over a period of hours.

The period of response coincides generally with that measured by other means. In particular, the results indicate that effects of the drug are present 1-12 hours after ingestion.

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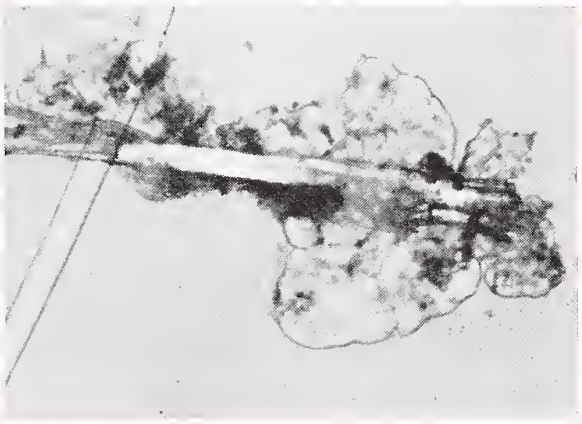
References

1. Russek, H.I.: Combined Vasodilator And Tranquilizer Therapy In Angina Pectoris: A Comparative Study With Statistical Analysis, Am. J. Med. Sciences, 249: 420, 1965.
2. Gensini, G.G., Di Giorgi, S., Murad-Netto, S., and Black, A.: Arteriographic Demonstration Of Coronary Artery Spasm And Its Release After The Use Of A Vasodilator In A Case Of Angina Pectoris And In The Experimental Animal, Angiology, 13: 550, 1962
3. Likoff, W., Kasparian, H., Lehman, J.S., and Segal, B.L.: Evaluation Of "Coronary Vasodilators" By Coronary Arteriography. Am. J. Cardiol., 13: 7, 1964.
4. Plotz, M.: Oral Sustained-Release Nitroglycerin Tablets In Angina Pectoris, Am. Pract. Dig. Treatment, 9: 903, 1958.
5. Corday, E.: Uncertainty About Nitroglycerin Effect Results In Electromagnetic Device. JAMA., 183: 27, 1963.
6. Blinder, S.: The Evaluation Of Controlled-Release Nitroglycerin Tablets (Nitrong) In The Treatment Of Angina Pectoris, Curr. Ther. Research, 7: 769, 1965.
7. Hedges, R.N., Jr., and Gordon, W.Z.: A Spectral Analysis Of Coronary Heart Disease, Angiology, 16: 728, 1965.
8. Charlier, R.: Coronary Vasodilators, Pergamon Press, 1961.
9. Rogers, W.J.H. and Turner, R.A.: Submitted for publication 1965.
10. Berry, J.W. and Roach, T.C.: An Evaluation Of Blood Nitrate Levels, Circulation, 17: 1041, 1958.

Trichomycosis Axillaris

Donald Rosman, M.D., *Los Angeles*



Trichomycosis Axillaris is a superficial infection of axillary or pubic hairs, characterized by firm nodules one-half the diameter of the hair composed of microorganisms. Red, yellow, black and fuscous varieties of the disease have been described and the variation in color probably is associated with specific differences in microorganisms present. The patient usually is asymptomatic and unaware of the infection.

Trichomycosis Axillaris is common in temperate as well as tropical areas. It is not limited to one sex or race, but the degree of sweating and personal hygiene probably alters the susceptibility. It is probable that the etiology varies with geographic location.

Crissey et al (1952) reviewed the literature and added the results of their own studies. They isolated a diptheroid organism from their cases, and suggested the name Corynebacterium Tenuis for this organism.

Castellani suggested that the etiologic agent is a nocardia, but there is little in the literature concerning the infection to indicate that a true nocardia is involved.

Based on Crissey's studies, the name Trichomycosis is a misnomer; however, until the etiology of the infection is critically studied in many parts of the world, a change in the name might be equally unsuitable.

Case Report

The patient, an adult white male, was examined by me February 11, 1966, because of an unusual condition of the right axillary hair. He had noticed that the hair in the area was rubbery and it would stretch when pulled. He also noticed hard concretions along the hair. The condition was completely asymptomatic.

On examination, I found white nodular concretions on the hair shafts in the right axilla. These concretions were about the size of a pin head and were firmly attached to the hair. The hairs fluoresced bright green under Wood's light.

The involved hairs were lighter in color than those in the left axilla and were lustreless. The hairs in the left axilla and the rest of the body were completely normal.

Shaving the hair in the right axilla and treating it with a one-quarter strength Whitfield ointment containing two per cent precipitated sulphur completely eradicated the condition.

One of the hairs was inoculated on Sabouraud's Dextrose Agar but there was no growth.

Summary

A case of Trichomycosis Axillaris of the right axilla is presented because of the extreme rarity of this condition in the Los Angeles area. Treatment for this condition is most effective and consists of shaving the hair and the use of a mild antifungal ointment containing 2-3 per cent precipitated Sulphur. Up to the present time there has been no recurrence of the condition.

3701 Stocker St.

References

1. Emmons, Binford and Utz: Medical Mycology.
2. Crissey, Rebel and Laskas: Studies on the Causative Organism of Trichomycosis Axillaris, Journal Investigative Dermatology, 19: 187-197, 1952.
3. Heldik and Smith: Fungus Diseases and Their Treatment.
4. Dubos, Rene J.: Bacterial and Mycotic Infections of Man.

APHORISMS and MEMORABILIA

ANDREW M. BABEY, M.D., *Las Cruces, New Mexico*

1. "It is suggested that most human beings know so little about how to live that unlimited leisure would mean disaster if not destruction. The business of leisure is part of the art of living, and the average man's sole notion of spending leisure is to be amused, preferably to have others hired to amuse him, or to rely on machines for his pleasures — the automobile, the slot machine, the airplane, the sports spectacle" —

"The Ambiguous Commodity" — E. P. Scarlett — *Archieves of Internal Medicine* — Vol. 117 (April) 1966, AMA pub. pp. 580-585.

2. "The practice of leisure is an ideal, an attitude of mind, and a philosophy which can be found only in a few individuals" — Ibid.

3. "Most people know what work is, what free time is, but fail to understand what leisure involves" — Ibid.

4. "It is clear that today there is teeming, vigorous life (whether the good life depends on your point of view), and precious little leisure" — Ibid.

5. "Lesiure is a state of being free of everyday necessity. It is not a free time, long weekends, months of vacation, or years of retirement. The activities (and leisure properly conceived is an active, not a passive, thing) of leisure are those in which the individual engages for their own sake, and at his own speed. Leisure presumes an education or some native instinct bred of the soil, an appreciation of certain things in life" — Ibid.

6. "Horace Bushnell once put the distinction in this way: work is activity for an end, leisure (play he called it, in sober Victorian fashion) is activity as an end. Leisure, I feel, has its spring in some fund of life back of the will and the business of life. In too many people that fund of life withers and is exhausted early in their career. Too many go on in the vain hope that if they drudge away today, there will be the compensation of leisure tomorrow. And so they wish away the best part of their lives, and presently find that the barely tolerable routine of labor is the only thing that keeps life from becoming intolerable" — Ibid.

7. "To attain leisure requires a stern act of the will. Leisure is compounded for a balance between detachment and involvement; it means securing, in the teeth of great odds, seasons of relaxation, getting as close to doing nothing as is possible for Western Aryan man to achieve, finding increasing delight in escaping from our servitude to Time, a grim determination to secure a free margin to one's life, a constant search for places where one may be away from the crowd and the bombardment of events" — Ibid.

8. "As James Thurber once said, 'It is better to have loafed and lost than never to have loafed at all.'" Ibid.

9. "Still in the field of paradoxes, it is a good rule to follow early in life whereby you make what you love best your avocation, not your vocation; or, by the same token, later in your career, to take up an avocation which gives you a deeper pleasure than your vocation" — Ibid.

10. "I doubt very much whether true leisure can be organized. For one thing, there is altogether too much organizing going on in these days. Community planning for leisure is almost a contradiction in terms" — Ibid.

11. "In one sense the dominating feature in the life of the educated man of today is his incessant battle with time. Too many things compete for his attention. As a bread-winner he must meet the demands of his work and the things he *has* to do; as a good citizen he must find time for the things he *ought* to do; as a man aware of the larger dimensions of life he must try to fit in the things he *wants* to do" — Ibid.

12. "No matter how widely our argument about leisure has ranged, it has inevitably returned to the individual. And that raises the question of the essential loneliness of man. We in North America, as in Britain, do not seem to be so obviously worried by the problems of loneliness" — Ibid.

13. "Sir Thomas Browne gave us the injunction: 'Be able to be alone.' We come at last to know that from without can come only "the counsel of a friend who is himself alone, and the whisperings of satraps, and the wind-borne incoherencies of the mob." And so it comes about that aloneness, one of the attributes of leisure, provides for each of us the citadel of our strength" — Ibid.

14. "Medicine has long passed from the stage of seeking to imbed large fragments of pattern recognition in the mind of the student; unfortunately, most medical textbooks have failed to recognize this change; they continue in a Linnean descriptive style more suited to nineteenth-century botany than to twentieth-century medicine" — H. A. F. Dudley — *The Lancet* — 12 March, 1966, pp. 589-90.

15. "A textbook writer seldom seems to emphasize that the principles outlined in one chapter may govern the happenings and descriptions in the next; indeed, it is uncommon for principles to be outlined at all. The absence of a thoroughly systematic basis, particularly in clinical medicine, has made most textbooks intellectually lifeless — mere works of reference" — Ibid.

16. "The planned obsolescence of the car manufacturer has yet to make a significant impact on medical textbooks. When, as in the case of a small minority of books, they are commercially successful the demand for new editions may not necessarily lead to adjustment of the writer's ideas to new information. Second and later editions seldom move from strength to strength; there is something depressing about the progress of a good book from bright adolescence through pompous middle age to disastrous senescence along with its author or editor" — Ibid.

17. "Perhaps the worst form of textbook is that farrowed by multiple authors; unless there is the most rigid editing to the point where the contribution is rewritten, they are nearly always choked by inconsistencies, packed with repetition, or rendered indigestible by variations in style" — Ibid.

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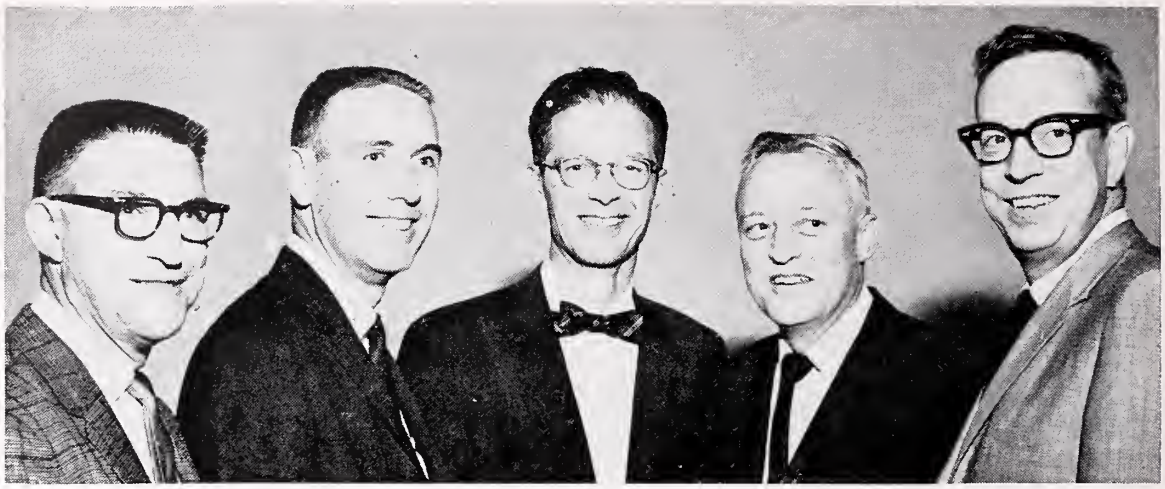

NEW OFFICERS—Dr. T. L. Carr, Albuquerque, center, was elected to succeed Dr. Robert P. Beaudette, Raton, as President of the New Mexico Medical Society at the 84th annual meeting of the Society in Albuquerque, May 9-13, 1966. New officers, left to right, are Dr. Earl Flanagan, Carlsbad, Vice-President; Dr. Emmit M. Jennings, Roswell, President-Elect; Dr. Carr; Dr. Beaudette; and Dr. John Abrums, Albuquerque, Secretary-Treasurer.

COMMUNITY SERVICE AWARD — Dr. Earl L. Malone, Roswell, right, is being congratulated by Dr. Robert P. Beaudette, Raton, Immediate Past-President of the New Mexico Medical Society, on having won the A. H. Robins Community Service Award. The Award was presented at the 84th Annual Meeting of the N. M. Medical Society in Albuquerque. Dr. Malone has been a General Practitioner in Roswell for 24 years and has been President of the N. M. Medical Society, the N. M. Chapter of the American Academy of General Practice, and was a delegate from the State Medical Society to the AMA from 1956 through 1965. Dr. Malone has been active in Community Health Chapters, Rotary, Chamber of Commerce, Roswell Symphony Association, the N. M. Medical Society Grievance Committee, Blue Shield and Boy Scouts.

Coming Meetings

Ninth Annual Ruidoso Summer Clinic sponsored by the New Mexico Chapters of the American Academy of General Practice and the American College of Obstetrics and Gynecology, Ruidoso, New Mexico, Chaparral Motel, July 25-30, 1966.

96th Annual Session of the Colorado Medical Society, Colorado Springs, The Broadmoor, September 25-28, 1966.

1966 Technicon International Symposium on Automation in Analytical Chemistry, New York City, Statler-Hilton Hotel, October 17-19, 1966.

Interim Meeting of the New Mexico Medical Society, Alamogordo, New Mexico, Holiday Inn, November 11-12, 1966.

85th Annual Meeting of the New Mexico Medical Society, Santa Fe, La Fonda Hotel, May 18-20, 1967.

48th Biennial Meeting of the Southwestern Medical Association, El Paso, Sheraton-El Paso Motor Inn, February 8-10, 1968.

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IN THIS ISSUE

VOL. 47, NO. 8

August, 1966

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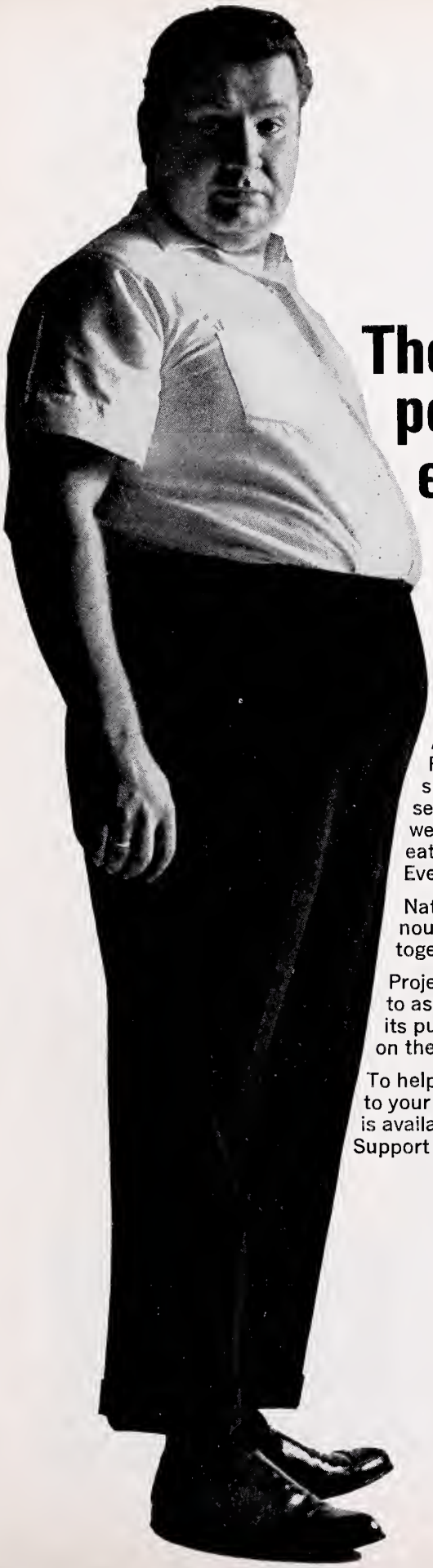

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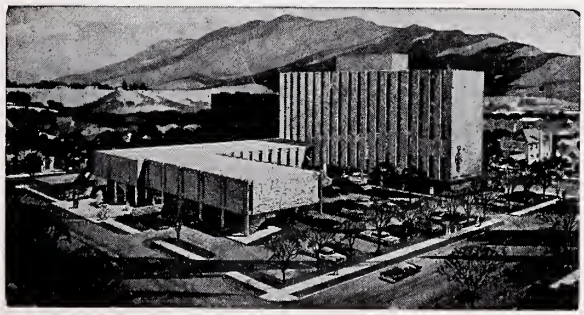

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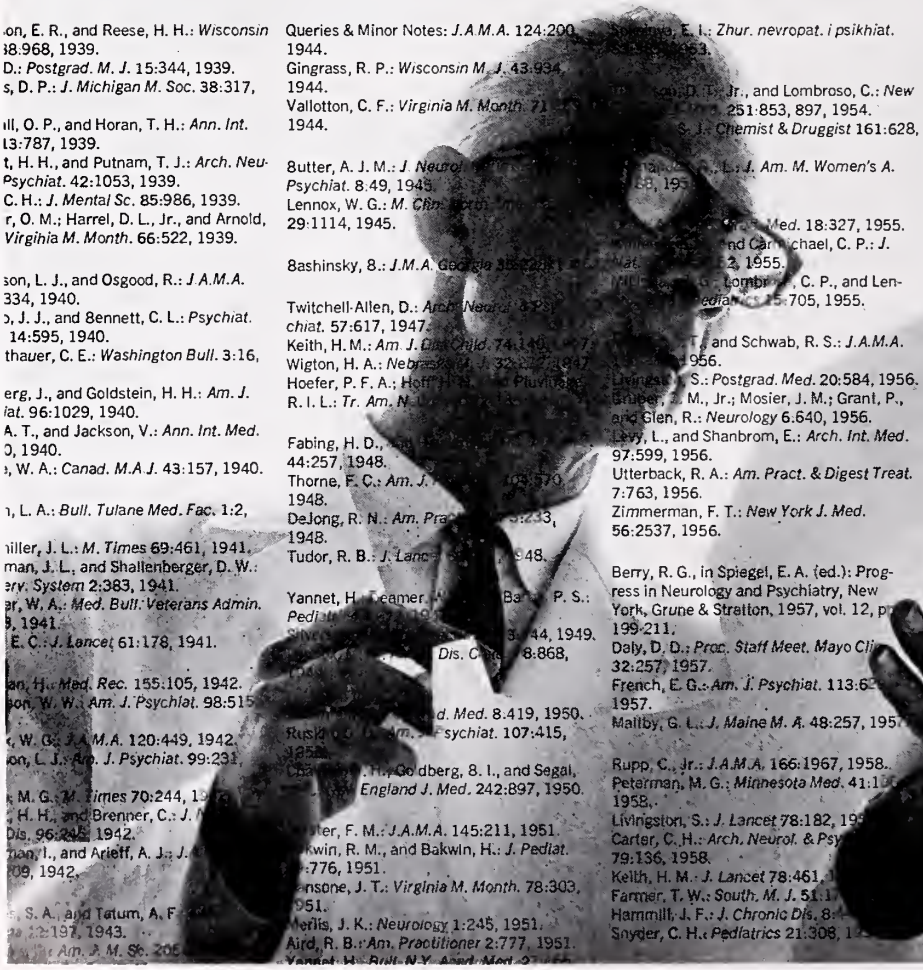

on, E. R., and Reese, H. H.: *Wisconsin* 38:968, 1939.
D.: *Postgrad. M. J.* 15:344, 1939.
s, D. P.: *J. Michigan M. Soc.* 38:317,

ill, O. P., and Horan, T. H.: *Ann. Int.* 13:787, 1939.
t, H. H., and Putnam, T. J.: *Arch. Neu-Psychiat.* 42:1053, 1939.
C. H.: *J. Mental Sc.* 85:986, 1939.
r, O. M.; Harrel, D. L., Jr., and Arnold, *Virginia M. Month.* 66:522, 1939.

son, L. J., and Osgood, R.: *J.A.M.A.* 334, 1940.
o, J. J., and Bennett, C. L.: *Psychiat.* 14:595, 1940.
thauer, C. E.: *Washington Bull.* 3:16,

erg, J., and Goldstein, H. H.: *Am. J. iat.* 96:1029, 1940.
A. T., and Jackson, V.: *Ann. Int. Med.* 0, 1940.
, W. A.: *Canad. M.A.J.* 43:157, 1940.

, L. A.: *Bull. Tulane Med. Fac.* 1:2,

iller, J. L.: *M. Times* 69:461, 1941.
man, J. L., and Shallenberger, D. W.: *ery. System* 2:383, 1941.
r, W. A.: *Med. Bull. Veterans Admin.* 3, 1941.
E. C.: *J. Lancet* 61:178, 1941.

an, H.: *Med. Rec.* 155:105, 1942.
son, W. W.: *Am. J. Psychiat.* 98:515
, W. G.: *J.A.M.A.* 120:449, 1942.
on, L. J.: *Am. J. Psychiat.* 99:231,
M. G.: *M. Times* 70:244, 1
H. H., and Brenner, C.: *J.*
Dis. 96:245, 1942.
an, I., and Arieff, A. J.: *J.*
09, 1942.
S. A., and Tatum, A. F.
2:197, 1943.
Am. J. M. Sc. 205

Queries & Minor Notes: *J.A.M.A.* 124:200, 1944.
Gingrass, R. P.: *Wisconsin M. J.* 43:934, 1944.
Vallotton, C. F.: *Virginia M. Month.* 71 1944.

Butter, A. J. M.: *J. Neurol. Psychiat.* 8:49, 1945.
Lennox, W. G.: *M. Clin. North.* 29:1114, 1945.

Bashinsky, B.: *J.M.A. Georgia* 35:

Twitchell-Allen, D.: *Arch. Neurol. Psychiat.* 57:617, 1947.
Keith, H. M.: *Am. J. Dis. Child.* 74:140,
Wigton, H. A.: *Nebraska M. J.* 32:
Hoefer, P. F. A.; Hoff
R. I. L.: *Tr. Am. N*

Fabing, H. D.,
44:257, 1948.
Thorne, F. C.: *Am. J.* 1948.
DeJong, R. N.: *Am. Pra* 1948.
Tudor, R. B.: *J. Lanc* 948.

Yannet, H. Teamer, Ba P. S.: *Pediat.* 44, 1949.
Dis. C 8:868,

d. Med. 8:419, 1950.
Rusk *Psychiat.* 107:415, 1950.
Goldberg, S. I., and Segal, *England J. Med.* 242:897, 1950.

ter, F. M.: *J.A.M.A.* 145:211, 1951.
Bakwin, R. M., and Bakwin, H.: *J. Pediat.* :776, 1951.
nsone, J. T.: *Virginia M. Month.* 78:303, 1951.
Merlis, J. K.: *Neurology* 1:245, 1951.
Aird, R. B.: *Am. Practitioner* 2:777, 1951.
Yannet, H.: *Bull. N.Y. Acad. Med.* 27

Jr., and Lombroso, C.: *New* 251:853, 897, 1954.
S. J.: *Chemist & Druggist* 161:628,

L.: *J. Am. M. Women's A.*

Med. 18:327, 1955.
nd Carmichael, C. P.: *J.* 2, 1955.
Lombr , C. P., and Len-*diat.* 15:705, 1955.

and Schwab, R. S.: *J.A.M.A.* 956.
S.: *Postgrad. Med.* 20:584, 1956.
M., Jr.; Mosier, J. M.; Grant, P., and Glen, R.: *Neurology* 6:640, 1956.
Levy, L., and Shanbrom, E.: *Arch. Int. Med.* 97:599, 1956.
Utterback, R. A.: *Am. Pract. & Digest Treat.* 7:763, 1956.
Zimmerman, F. T.: *New York J. Med.* 56:2537, 1956.

Berry, R. G., in Spiegel, E. A. (ed.): Progress in Neurology and Psychiatry, New York, Grune & Stratton, 1957, vol. 12, p 199-211.
Daly, D. D.: *Proc. Staff Meet. Mayo Cli* 32:257, 1957.
French, E. G.: *Am. J. Psychiat.* 113:6 1957.
Mallby, G. L.: *J. Maine M. A.* 48:257, 195

Rupp, C., Jr.: *J.A.M.A.* 166:1967, 1958.
Peterman, M. G.: *Minnesota Med.* 41:1 1958.
Livingston, S.: *J. Lancet* 78:182, 19
Carter, C. H.: *Arch. Neurol. & Psy* 79:136, 1958.
Keith, H. M.: *J. Lancet* 78:461,
Farmer, T. W.: *South. M. J.* 51:1
Hammill, J. F.: *J. Chronic Dis.* 8:
Snyder, C. H.: *Pediatrics* 21:308, 1

Yahr, M. D., and Merritt, H. H.: *In Neurol.* 1:76, 1959.
Thomas, M. H.: *J.A.M.A.* 169:457
Peterman, M. G., and Thomas, E. *Times* 87:1044, 1959.
Bray, P. F.: *Pediatrics* 23:151, 19
Mulder, D. W.; Daly, D. D.; Rushto and Yoss, R. E.: *Postgrad. Med.* 2 1959.
Forster, F. M.: *Wisconsin M. J.* 58 1959.
Robertson, E. G.: *Postgrad. Med.* 1959.

Schwade, E. D.: *Geriatrics* 15:11,
Schade, G. H., and Gofman, H.: *P* 25:151, 1960.
Forster, F. M.: *Postgrad. Med.* 27 1960.
Livingston, S.: *J.A.M.A.* 174:135,
Friedlander, W. J.: *Am. J. Psychia* 1960.

Kane, C. A.: *Boston Med. Quart.* 1961.
Holowach, J.; Renda, Y. A., and W *J. Pediat.* 59:339, 1961.
Baker, A. B.: *J. Lancet* 81:235, 1
Neurology 11:912,

; Brock, J. T., ar *& Therap.* 3:
Neurol. 3:18

M. J. 12:49
Dis. Pediat. 2:233
Clin. North Am

New England J. Med 1963.
; Winters, W.; Kokenge, R ell, F.: *Arch. Neurol.* 11:6
er, R. D.: *GP* 30:125, 1964.
D.; Sexton, J. A., and Davis *diat.* 64:499, 1964.

ingston, S.: *Children* 12:9, 196
Naughton, F. L., and Lloyd-Sm

Symposium on Tropical Diseases

William Beaumont General Hospital, El Paso

(Editor's Note: This is the first of three parts of a Symposium on Tropical Diseases presented at William Beaumont General Hospital in El Paso, Texas. The second and third parts will be published in subsequent issues of SOUTHWESTERN MEDICINE.)

MALARIA *

Lt. Col. Robert H. Moser, MC, *Chief, Department of Medicine*

Throughout the history of warfare one can follow the heavy imprint of malaria.[1] This disease has been known on this earth for over 2,000 years. Over one billion people still live in areas where malaria flourishes. Four million dwell where no malarial eradication program exists. Malaria is a disease with 300 million sufferers and takes the lives of 3 million people each year. It has been doing this for centuries. And, again, as it has countless times in the past, it is influencing the fortunes of war. In South Viet Nam malaria has become a factor of vast military significance.

The incredible capability of malaria to destroy the power of armies is legendary. In World War I during the Macedonian campaigns of 1916, British, French, and Bulgarian armies were decimated to the point of total immobility. The stalemate persisted for three years; in the British Macedonian Forces alone, 163,000 out of 435,000 men were hospitalized. The other combatants fared little better.

In World War II in the South Pacific Theater of Operations malaria (primarily vivax) caused five times as many casualties as combat operations. In September 1943, the malarial attack rate was 700 per 1,000 men. In New Guinea the Australian 7th Division reported malaria to be the cause of 90 per cent of casualties due to disease, and 82 per cent of total casualties. This was not a unique experience. In the last days of fighting on Bataan 20,000 American and Filipino soldiers were infected with malaria. In the United States Army alone in the early years of World War II, 500,000 cases of malaria were reported. This caused General Douglas MacArthur in May 1943, to say "this will be a long war if for every division I have facing the enemy I must count on a second in the hospital with malaria and a third division convalescing from this debilitating disease."[2]

I have the feeling that General Westmoreland is having similar thoughts.

Nor is this exclusively a military problem; through the modern means of jet transportation, any physician may be confronted with a patient who returns from an endemic area within the incubation period or during the period when suppressive therapy is effective (9-12 days).[3]

The immediate problem is one of dreadful simplicity. In recent years we have come upon the revelation that there are certain strains of falciparum which do not respond to chloroquine. This remarkable drug was developed in World War II, and for 18 years it was singularly successful in controlling clinical manifestations of falciparum malaria. But in 1960, for the first time, resistant falciparum strains were reported from Colombia, Brazil, and Southeast Asia. Over 90 per cent of the identifiable malaria being encountered in

*This section was presented at the Loma Linda Post-Graduate Convention in Los Angeles on March 15, 1966.

Southeast Asia is due to *Plasmodium Falciparum.*

In 1962 the first American military casualty of the resistant falciparum malaria was a Marine at Nha Trang. Subsequently, this particular strain was transmitted to human volunteers at Statesville, Illinois, and became known as the Snell strain. It proved resistant to all forms of antimalaria therapy except pyrimethamine* and quinine. To achieve parasite destruction doses of quinine were required that consistently caused cinchonism.[3]

In Cambodia, six out of eight members of the 1963 Owle's expedition from Kuala Lampur developed falciparum malaria despite what had been considered adequate prophylaxis.[3]

In the same year, in areas of Thailand and upper Malaya, additional chloroquine-resistant strains were discovered. British Commonwealth troops in upper Malaya succumbed to this illness in significant numbers.[3]

In the Fall of 1965, the first impact of chloroquine-resistant malaria as a factor of tactical significance, struck American forces throughout South Viet Nam. Prior to this time only sporadic cases had been seen. When the First Cavalry Division moved into the South Central highlands around Plei Me and became involved in the now famous eight day battle in the Ia Trang Valley, large number of malaria casualties began to arrive at hospital units soon after the combat casualties had been sorted and treated. Suppressive therapy delayed the onset of clinical signs and symptoms for 19 days.[3]

Even the veteran Korean Tiger Division which shared the field of combat with American and Australian troops, developed falciparum malaria casualties. These troops had come from areas where vivax malaria was endemic. It had been assumed that they had some degree of immunity; but this was erroneous. Even the natives of South Viet Nam and the native RVN troops had difficulties with falciparum malaria. There have been indications that the Viet Cong may have suffered significant losses to malaria as well.[3]

The military aspects of the problem are complicated by the fact that the incidence of malaria among troops on active combat or patrol duty is several times higher than that among support troops based in the same area. This indicates that personal and environmental control measures are still effective to the extent that the tactical situation will permit. Troops engaged in direct combat operations have less inclination or time to use insect repellents or protective clothing. At present, vector control is limited to areas that are "secure."[4]

Clinical Manifestations and Life Cycle

The clinical appearance of falciparum malaria frequently does not conform to the classic format of paroxysmal chills, fever, and diaphoresis. Patients have been seen whose principal complaints are intestinal distress, musculoskeletal pain, postural hypotension, unexplained high fever, irrational behavior, and delirium. It has become axiomatic that malaria should be considered in the differential diagnosis of any individual with a fever which cannot be readily explained, who has recently been in an endemic area. Perhaps one could include scrub typhus and leptospirosis in this category, but malaria is the foremost consideration. Needless to say, early diagnosis and swift, decisive therapy are extremely important.

Routine thin blood smears are less apt to show parasites in falciparum malaria than in other varieties. Thick smears are mandatory. Each smear must be thoroughly examined by someone trained in parasite recognition for perhaps 10-15 minutes before the preparation is declared negative.

Before discussing current management it might be well to review briefly the somewhat complicated life cycle of the *Plasmodium falciparum.* There are two principle cycles.

The sexual cycle occurs within the mosquito. We will not be concerned with this except to say that certain anti-malarial agents affect the parasite within the mosquito. These are called sporonticides. The other, asexual cycle, occurs within the animal host. When the mosquito inserts its proboscis into a blood vessel, sporozoites are injected. These migrate from the vascular compartment within 30-40 minutes without parasitizing red cells to enter the parenchymal cells of the liver. Here the parasites undergo asexual multiplication. It is highly significant from clinical and therapeutic aspects that *Plasmodium falciparum* undergoes only one tissue or exoerythrocytic phase in the liver before re-entering the blood stream and parasitizing red blood cells. *Plasmodium falciparum* never re-enters the tissue phase. Therefore, drugs which destroy the intra-erythrocytic parasite

*Daraprim

— the blood schizont — should be curative and not merely suppressive. This single cycle is not the situation in other forms of malaria; they all have repeated tissue-erythrocyte cycles.[5]

However, in falciparum malaria there have been rare cases of relapses occurring up to one year following treatment. The explanation is not completely understood.

Parasitization of the red cells by *Plasmodium falciparum* occurs rapidly and voraciously after the tissue phase. Prognosis is extremely grave when one can count 500,000 parasites per cubic milliliter, or when about 10-12 per cent of the total red blood cells contain the parasites. Fatalities have occurred with parasitism of 25,000 per cubic millimeter erythrocytes (0.5%). Intravenous quinine is recommended when parasitism reaches three per cent.[4]

Anti-malarial drugs that destroy asexual erythrocytic forms are termed blood schizonticides. These are suppressive drugs; chloroquine is the prime example. Suppression refers to the prevention or elimination of symptoms, and this is related to destruction of the schizonts in parasitized red cells, to prevent hemolysis and its sequelae. Suppressive therapy may occasionally eliminate the tissue phase parasite. Primaquine is especially effective in this instance.[5]

The evolution of clinical symptoms is due to the rupture of parasitized erythrocytes at the end of the schizont stage. Upon hemolysis of the red cell, merozoites are released which in turn parasitize new red blood cells. It has been assumed that rupture of erythrocytes, with liberation of products of metabolism of both parasites and red cell, discharges this toxic material into the blood. It is proposed that this may be the mechanism of the malarial paroxysm. Synchronous rupture on a rhythmic basis is seen in all forms of malaria except falciparum. Asynchronous rupture of parasitized red cells accounts for its many clinical disguises. A few of the liberated merozoites reinfect the erythrocytes and become sexual forms, either male microgametocytes or female macrogametocytes. Agents effective against schizonts do not ordinarily destroy gametocytes. It is in this form that the parasite is withdrawn from the blood stream by the female Anopheles mosquito to complete the life cycle. It is clinically significant that merozoites of *Plasmodium vivax* attack reticulocytes, which tends to limit the parasitemia. On the other hand, merozoites of *Plasmodium malaria* attack aging erythrocytes, again, a limiting factor. However, *Plasmodium falciparum* produces merozoites which attack erythrocytes without regard to age and it is this characteristic which makes *plasmodium falciparum* the most dangerous of all malarias. Although gametocytes usually do not survive in the host more than a few weeks after treatment, it is possible for the clinically "cured" patient to transmit the disease during this interval. There are instances of some asymptomatic immune carriers who may carry the gametocytes for months.

Treatment

It has been found that many of the southeast Asian strains are not only resistant to chloroquine and other 4 aminoquinilones and mepacrine,* but also to newer anti-malarials such as chloroguanide and pyrimethamine. Response to quinine is still adequate in most cases, but there are exceptions. The problem is critical because our stock pile of quinine is not unlimited, and sources of supply are very restricted.

A parallel situation existed in World War II when the Japanese moved rapidly into the uplands of Java and seized the few thousand acres of land bearing cinchona trees. This gave the Japanese a virtual monopoly on the only means which man had known for 300 years for fighting malaria. The Germans suffered similar quinine deprivation in World War I which stimulated an intensive search for synthetic substitutes for quinine. With the advent of the 8 and later the 4 aminoquinolones and other powerful drugs, the need to maintain cinchona tree plantations diminished. Interest in the development of new anti-malarial drugs also decreased sharply. Now, at a time when quinine seems to be the only effective medication in certain instances, we may find a significant shortage on our hands.

Prophylaxis

Current prophylactic measures are undergoing continuous analysis and re-evaluation. At the present time a weekly tablet containing chloroquine (a blood schizonticide) 300mgm base and primaquine (a tissue schizonticide) 45mgm base is given once a week. A control study is being done in which other soldiers are taking DDS (diaminodiphenyl-sulfone) the old antileprosy drug, 25mgm every day in addition to weekly chloroquine-

*Atabrine

primaquine.

Prophylactic quinine has been suggested but is not entirely practical. A half of a gram of quinine daily will still permit a 50 per cent breakthrough incidence of malaria, especially in subjects undergoing extreme exertion.[6] In addition, there simply is not sufficient quinine available in stockpiles for a broad prophylactic program. Even if this were feasible, at the 0.5 gm level a good many people will have discomfort and tinnitus which will impair their military effectiveness, and may last for years.

Medical Management

Problems presented by the current falciparum strains in South Viet Nam are related primarily to chloroquine resistance and the consequences of uninhibited parasitism of erythrocytes.

There have been some deaths in South Viet Nam directly attributed to falciparum malaria. The number may seem inconsequential, but one must recall that only 302 deaths directly attributed to malaria were recorded among all the malaria victims among U. S. Troops in World War II.

By current estimates about 20-50 per cent of patients with falciparum malaria will respond to "first echelon treatment" with chloroquine phosphate 1.0 gm (2 tablets, each containing 300 mgm of base) and 0.5 gm six hours later and then 0.5 gm once daily for the next two days. This is a total dose of 2.5 gm of chloroquine phosphate in three days. If the patient is unable to take chloroquine by mouth the drug may be given as the dihydrochloride, 250 mg intramuscularly repeated at six hour intervals. It is advisable to switch to the oral preparation as soon as possible. This "first echelon" program is advised for all patients with any type of malaria who are not critically ill and who have not received a previous course of chloroquine. However, with this therapy, 80 per cent of patients encountered in RVN suffered clinical relapse in from five days to four weeks.[4]

The recommended therapy changes drastically for patients who are extremely ill, those who have not responded to chloroquine treatment in the past, or those who have a clinical relapse despite a previous course of chloroquine. The drug of choice in this situation is quinine alone or in combination. Several programs incorporating quinine in combination with other drugs have been advocated. The most successful has been:

Quinine sulfate 600 mgm q8h x 10, and
Sulfadiazine, 2.1 gm x 10, and
Pyrimethamine, 75 mgm q.d. x 3.[4]

Of 60 patients treated with this combination, none relapsed over an observation period of 30 days. Relapses have occurred with chloroquine and quinine. Other regimens utilizing quinine, DDS and pyrimethamine in varying combinations have been somewhat less successful. Some patients tend to develop postural hypotension when quinine is given in this dosage. They should be kept at bed rest during the period of treatment. Urine output should be monitored. If oliguria develops quinine should be discontinued. During periods of oliguria, quinine blood levels rise precipitously and acute cinchonism may ensue. In the severely ill or chloroquine-refractory patient who is unable to retain quinine orally, the drug may be given intravenously. It is indicated under the following circumstances:

a) Whenever hiccups, nausea or vomiting, intestinal colicky pain, diarrhea, epistaxis, petechial hemorrhage, hyperpyrexia, collapse, delirium or coma are present.

b) Whenever three per cent or more of red blood cells are parasitized or if the patient remains quite ill or still shows positive smears at the end of the third day after the start of treatment.[4]

Oral quinine, of course, should be resumed as soon as practicable. Quinine dihydrochloride 600 mg in 200 cc of normal saline should be given by slow intravenous drip over 30 minutes with careful observation of blood pressure and heart rate to insure early detection of hypotension or arrhythmias. This dose is repeated at eight hour intervals for up to 10 days. Oral therapy, by stomach tube, if necessary, should be instituted as soon as possible.

DDS had a clinical trial in northeastern Tanzania; it demonstrated an ability to potentiate pyrimethamine.[7] A new long-acting sulfanomide preparation, sulfa-orthodimethoxine or Fanasil has been tried in Tanganika.[8] It was used against pyrimethamine-resistant falciparum malaria. This sulfa-orthodimethoxine was an effective schizonticide when given in doses of 500 mg a week alone; the addition of pyrimethamine 25 mg a week contributed nothing.

Patients treated for malaria by any technique must be observed for four weeks following completion of treatment by weekly thick blood smears. In addition, the patient should be placed on the chloroquine-primaquine prophylactic tablet at weekly intervals for a total of six to eight weeks as further insurance against the late emergency of *Plasmodium vivax*. This tablet should never be used in the management of an acute malarial attack since the amount of primaquine may induce hemolytic reactions. The primaquine in this instance is used to destroy the exoerythrocytic or tissue form of vivax malaria as stated earlier.

Clinical trials have been carried out with a repository antimalarial agent cycloguanine pamoate. Results have been disappointing. The drug was studied in villages in East Pakistan and it was noted that one to two per cent of patients developed sterile abscesses at injection sites. Duration of protection with cycloguanine pamoate was less than six months.[6]

There are several classic complications of severe falciparum malaria. Perhaps the most malevolent is Black Water Fever which may follow violent hemolysis. You will recall that the falciparum attacks erythrocytes of all ages and is capable of parasitizing up to 12 per cent of the total red cell population. This results in rapid intravascular hemolysis which inundates the reticuloendothelial system with hemoglobin. This is followed rapidly by hemoglobinemia, hemoglobinuria, anemia, sludging, and oliguria with renal shut-down. This in turn causes azotemia. Water loading superimposed on water retention can lead to cardiac decompensation and possibly death. Hyperkalemia is an additional threat during renal shut-down. Treatment is the same as for an acute hemolytic transfusion reaction. Intravenous mannitol is given rapidly in 5-10 minutes after the patient has been sufficiently hydrated. Urine volume within the next two hours must exceed 60 ml per hour or fluids should be restricted. If urine flow is below 60 ml per hour, the patient should be regarded as suffering acute renal failure, and fluids appropriately restricted. Expectant management is advisable employing replacement fluids, resins against hyperkalemia and possibly peritoneal or extracorporeal dialysis. If urine flow is adequate, hydration should be continued and mannitol administered often enough to maintain urine flow at 100 ml per hour or more. During the massive infusion of mannitol it is possible that the patient may become hyponatremic; this should be anticipated and treated appropriately.

In addition, intravenous heparin has been advocated to decrease blood viscosity and prevent sludging. Mannitol also seems to have some protective effect against sludging while providing the osmotic diuretic force for increased renal perfusion. Occasionally packed red cells may be advisable to combat the severe anemia. A complication may arise if one is obliged to give quinine while the patient is oliguric. As indicated previously, quinine levels can approach toxic proportions in the presence of oliguria. Consequently, periodic blood quinine levels would be ideal, but if this is not available, blood urea nitrogen assay should be done. In addition, it is advisable to monitor central venous pressure; over-hydration should be avoided.

A problem that has arisen in certain cases of falciparum malaria is significant water retention. These patients suffer excessive thirst which apparently is not auto-regulated; it can result in significant water overloading. Some patients have gained from 8-15 lbs. over a very brief period of time.[3]

An additional complicating feature has been diarrhea associated with malabsorption which may result in hypoalbuminemia. This phenomenon when associated with excessive water ingestion has aggravated over-hydration. Occasionally this has resulted in cerebral edema which may be confused with falciparum cerebral malaria. Consequently parenteral fluids should be used with caution in the patient who presents the unusual combination of excessive thirst and impending oliguria. The problem of intracerebral edema has been attacked successfully by the use of intravenous dexamethasone.

Cerebral malaria as a complication of *Plasmodium falciparum* infection is a comparatively rare, but very serious and sometimes fatal complication (2.3% in one series of 6,000 patients), but has been a cause of death in only a few patients in Viet Nam. It is due to sludging of parasitized erythrocytes in cerebral vessels. Any neurologic and/or psychiatric syndromes may be simulated. The highly motivated combat soldier who ignores headache, malaise, and fever for a few days may lapse into coma and die quickly.

At least one death has been related to pulmonary insufficiency due to intravascular sludging, anemia,

and anoxia in pulmonary vessels.[3]

Immunization

Powell and Brewer[9] have been investigating the possibility of active immunization against malaria. Most of the experimental work on immunity has centered on the earliest erythrocytic form of the parasite, the trophozoite.

Any immunity that is acquired by the host is directed chiefly against the trophozoite. Efforts to produce acquired immunity artificially with suspensions containing killed or inactivated parasites have not been rewarding in human malaria. Some positive results have been obtained with avian malaria. It is considered possible that significant protection against mosquito-induced infections may be afforded in humans by inoculation of a vaccine containing inactivated sporozoites emulsified in mineral oil. In any event, such immunity will probably be only strain specific. The ultimate goal of creating anti-malarial vaccines tailored to incorporate several strains endemic to a specific geographic area is possible. Investigation in all these areas is continuing.

Vector Eradication

The advent of DDT, malathione, and other effective insecticides was heralded by many as the probable answer to malaria control on an international scale. In 1961 an enthusiastic malarial eradication program was launched by the World Health Organization in Viet Nam. It was enjoying remarkable success, but it came upon evil times as the Viet Cong began to systematically eliminate malarial control people three years ago as a political expedient.[3] At the present time there is no practical program for widespread vector control in Viet Nam until the military situation becomes stabilized. The critical area remains the South Central highlands where the enemy is still quite active.

The females of certain species of Anopheles mosquitos serve as the arthropod vector for human malaria. Of the approximately 200 known species of Anopheles mosquito, only about 60 have been incriminated as vectors. In certain areas there may be more than one vector species, but usually a single species assumes the dominant role of villain.

The Anopheles is diabolically equipped for its role in the spread of malaria. Unlike the common household mosquito, Anopheles approaches silently, bites inoffensively, and escapes undetected. The problem has been further complicated by the realization that there are some new mosquito vectors involved. For example, in Thailand it was believed that Anopheles minimus was the only vector. Recently it has been discovered that Anopheles balabacensis will also carry malaria. When one realizes that balabacensis is a deep jungle breeder, languishing in the shade under broad jungle leaves, has a flight range of one to two kilometers, comes into dwellings late at night, and does not light upon the DDT-sprayed walls, one gets some idea of the problems involved in controlling such a vector.[6]

Colonel William D. Tigertt, Commanding Officer of Walter Reed Army Institute of Research, and an outstanding authority on malaria, has expressed the frustration of this experience by saying—only half-facetiously—that in order to control malaria in jungle areas, the only solution would be to move the jungle.[6]

Conclusion

In coordination with the Armed Forces Epidemiology Board's Commission on Malaria a tremendous research effort is under way. Some investigators are studying the structure of the plasmodia in an effort to devise new ways to attack the parasite. Others are involved in systematic chemotherapeutic evaluation programs. The biochemists are directing their attention to better methods of assaying anti-malarial drug levels in body fluids. The association of hypo or ahaptoglobinemia in certain areas where *Plasmodium falciparum* abounds has prompted speculation and investigation on the relationship between the two. Work in vector control and in improving skin repellants continues. Time does not permit a detailed discussion of this multi-faceted, intensive, investigative effort, but it can be stated quite frankly that this represents the most dramatic challenge to military medical research since the end of World War II.[6]

It may be assumed that malaria will remain a public health menace for many years to come, aside from the immediate military problem in Viet Nam. We have learned from the chloroquine experience that we cannot relax our vigil in the search for effective means of mosquito eradication and anti-plasmodial chemotherapy. The continuing lack of political and military stability in areas of the world where malaria remains a vast endemic

problem will persist as an obstacle to the ultimate elimination of malaria as a major disease of man. Perhaps when the last bugle blows and the free men of this earth can arise to resume the tasks of peace—we may be able to conquer once and for all this ancient and implacable enemy.

LEPTOSPIROSIS

Leptospirosis has undergone a significant conceptual evolution since it was first popularized in 1935. In the past it was customary to identify clinical signs and symptoms on the basis of specific leptospiral strains. It is now known that almost any strain can produce illness of almost any degree of severity. Thus Weil's disease is no longer the clinical syndrome seen exclusively with *Leptospira ictero-hemorrhagica,* but rather represents the severest form of leptospiral infection which is attended by fever, hemorrhage, and hepato-renal involvement; this syndrome can develop with any strain.

The classic clinical configuration of a leptospiral infection consists of a biphasic illness. There may be a prodromal interval of malaise; this is soon followed by a five to seven day period of sudden fever, chills, violent headache, myalgia, purpura, conjunctivitis, and gastrointestinal symptoms. This is called the "septicemic" stage and during this first week the organism may be recovered from blood and spinal fluid. Thereafter it will not be seen in these areas. There follows a two to three day interval of defervescence with clearing of clinical signs and symptoms. In mild "anicteric" cases this may conclude the illness. In more severe "icteric" cases (about one out of every three or four), the "immune" phase will follow.

This is characterized by a secondary rise in temperature (not as high as in Stage I) with development of an aseptic meningitis. No leptospira will be found in the cerebrospinal fluid, but pleocytosis is common. Early, the predominant cells may be neutrophils; by the 12th day lymphocytes will make up 80-90 per cent of the cells. Counts up to 500/mm^3 have been recorded. Protein elevation to 140/mg/100 ml has been seen, but normal spinal fluid glucose levels are usual. Curiously, bile may be seen in the spinal fluid in icteric cases of Weil's Disease. Meningitis is the most prominent clinical manifestation, but the 20-40 per cent mortality that has been described in Weil's Disease is related to severe hepatorenal failure with hemorrhage. Uveitis may follow the conjunctivitis; occasionally corneal opacifications may be found as a late sequel.

Diagnostic confirmation can be made in Stage I by demonstration of the leptospira in blood or spinal fluid. Occasionally, muscle biopsy (gastrocnemius) may be helpful after the first week. In Stage II diagnosis can be established by discovering the organism in urine or aqueous humor. Serologic evidence in Stage II can be obtained by the complement fixation test if the titre is 1:40 or higher (or a rise is demonstrated), or by "agglutination-lysis" test, if a titre of 1:400 or higher is demonstrated.

Management is symptomatic. Claims for effectiveness of antibiotics have not been confirmed. Supportive management (including dialysis in the event of renal shutdown) is mandatory.

Leptospirosis should be considered in the differential diagnosis of aseptic meningitis, fever with jaundice, hepatorenal syndrome, severe conjunctivitis and/or uveitis, and febrile diseases associated with muscle pain and tenderness.

References

1. Moser, R. H.: Of Plagues and Pennants. Mil. Review. *45:*71-84, 1965.
2. Blount, R. E.: Malaria — An Unsolved Military Medical Problem, Amer. J. Med. Sc. *247:*407-411, 1964.
3. Blount, R. E.: Personal Communication, January 1966.
4. Blount, R. E.: Report to The Surgeon General, "Malaria in Vietnam." 29 January 1966.
5. Powell, R. D.: The Chemotherapy of Malaria, Clin. Pharm. & Therap. *7:*48-76, 1966.
6. Armed Forces Epidemiology Board: Minutes of the Open Session. 7, 8 Dec. 1964.
7. Taing, A. B. G.: Treatment of Acute Falciparum Malaria with Diaphenylsulfone in Northeast Tangeria. Tr. Roy. Soc. Trop. Med. Hyg. *59:*251-253, 1965
8. Taing, A. B. G.: Antimalarial Effect Sulfaorthodimethoxine (Fanasil) Brit. Med. J. *2:*1439-1440, 1964.
9. Powell, R. D. and Brewer, G. J.: Active Immunization Against Malaria. Am. J. of Trop. Med. Hygiene. *13:*228-232, 1964.

Coming Meetings

American Physiological Society, Houston, Baylor University, August 29-September 2, 1966.

Flying Physicians Association, Las Vegas, Nevada, The Dunes Hotel and Country Club, September 11-16, 1966.

96th Annual Session of The Colorado Medical Society, Colorado Springs, The Broadmoor, September 25-28, 1966.

Texas Dermatological Society, El Paso, October 1-2, 1966.

Western Orthopedic Association, Tucson, Ramada Inn, October 2-6, 1966.

American Society of Plastic and Reconstructive Surgeons, Las Vegas, Nevada, Flamingo Hotel, October 2-7, 1966.

Medical Society of the United States and Mexico, Tucson, Pioneer Hotel, October 5-7, 1966.

American College of Surgeons, San Francisco, Fairmont Hotel, October 10-14, 1966.

American College of Obstetricians and Gynecologists, Houston, October 24-26, 1966.

American Association of Public Health Physicians, San Francisco, October 31-November 4, 1966.

American Association for Automotive Medicine, Alamogordo, New Mexico, Cloudcroft Hotel, November 10-11, 1966.

Interim Meeting of the New Mexico Medical Society, Alamogordo, New Mexico, Holiday Inn, November 11-12, 1966.

Western Surgical Association, Phoenix, Del Webb's Townehouse, November 16-19, 1966.

Southwest Allergy Forum, Galveston, Flagship Hotel, January 19-21, 1967.

American College of Surgeons, Arizona Chapter, Tucson, Arizona Inn, January 20-21, 1967.

TMA District 1 Meeting, Pecos, February 4, 1967.

American Academy of Pediatrics, San Francisco, Hilton Hotel, April 3-4, 1967.

American Society of Internal Medicine, San Francisco, St. Francis Hotel, April 7-9, 1967.

Southwestern Surgical Congress, Phoenix, Del Webb's Townehouse, April 10-13, 1967.

American College of Physicians, San Francisco, Fairmont Hotel, April 10-14, 1967.

American Academy of Neurology, San Francisco, Hilton Hotel, April 24-29, 1967.

Arizona Medical Association, Phoenix, Townehouse, April 26-29, 1967.

American Gynecological Society, Phoenix, Arizona Biltmore Hotel, May 4-6, 1967.

Texas Medical Association, Dallas, May 4-7, 1967.

85th Annual Meeting of the New Mexico Medical Society, Santa Fe, La Fonda Hotel, May 18-20, 1967.

48th Biennial Meeting of the Southwestern Medical Association, El Paso, Sheraton-El Paso Motor Inn, February 8-10, 1968.

Courses Offered in Ophthalmology

The Institute of Ophthalmology of the Americas is offering the ninth series of post-graduate courses for specialists in Ophthalmology. They will be given from September 7 through November 19, 1966. The courses will include the following: Advances in Ocular Prostheses, Biomicroscopy, Cataract Diseases, Clinical Bacteriology, Contact Lenses, Corneal Diseases, Electrophysiology and Applied Physiology of the Eye, Enucleation and Evisceration, Glaucoma, Lacrimal Sac Surgery, Low Vision Aids, Motor Anomalies, Ocular Geriatrics, Ocular Neuro-Ophthalmology, Ocular Radiology, Ocular Therapeutics, Ocular Trauma, Ophthalmoscopy, Pathology, Pediatric Ophthalmology, Perimetry, Physiologic Optics, Plastic Eye Surgery, Radioisotopes in Ophthalmology, Refraction, Retinoscopy, Retinal Detachment, and Surgery of the Orbit and Uveitis. At the conclusion of the regular series, there will be a one-week review course in basic sciences in Ophthalmology.

For catalogue and additional information write: Jane Stark, Registrar, The Institute of Ophthalmology of the Americas, The New York Eye and Ear Infirmary, 218 Second Avenue, New York, New York 10003.

Medical Writers to Meet

"The Changing World of Medical Communication" will be the theme of the American Medical Writers' Association annual meeting to be held at the Waldorf-Astoria Hotel in New York City, September 29 through October 2, 1966.

A varied program of panel discussions, round tables, workshops, and plenary sessions is being organized by the Program Committee under the chairmanship of Alexander B. Gutman, M.D., and co-chairmanship of Martha Dana. Alvina Rich Lewis, secretary-treasurer of the A.M.W.A., is

serving as chairman of the Local Arrangements Committee, which has planned a variety of social events. Registration is $15 for non-members.

For further information write: James E. Bryan, Executive Secretary, American Medical Writers' Association, 2000 P Street, N.W., Washington, D.C. 20036.

AMA Sponsors Conference on Sports

The Eighth National Conference on the Medical Aspects of Sports, sponsored by the American Medical Association under the auspices of its Committee on the Medical Aspects of Sports, will be held in Las Vegas, Nevada, at Caesar's Palace on November 27, 1966. The conference is held annually in conjunction with and on the first day of the Clinical Convention of the AMA.

The conference will cover a wide range of subjects of interest to those serving school and college athletic programs. Included will be forums and discussion sections relating to criteria for immediate management of knee injuries, resources for grass roots supervision of sports, medical preparations for international competitions, and the relationship of athletic fitness to physical fitness.

The conference is open to key nonmedical athletic personnel as well as to interested physicians. For further information write: Secretary, Committee on the Medical Aspects of Sports, American Medical Association, 535 North Dearborn Street, Chicago, Illinois 60610.

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IN THIS ISSUE

VOL. 47, NO. 9

September, 1966

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*Neyroud, M.: Praxis 44:648-650 (July 14) 1955.

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*Schiller, I. W., and Lowell, F. C.: New England J. Med. 261:478, 1959.

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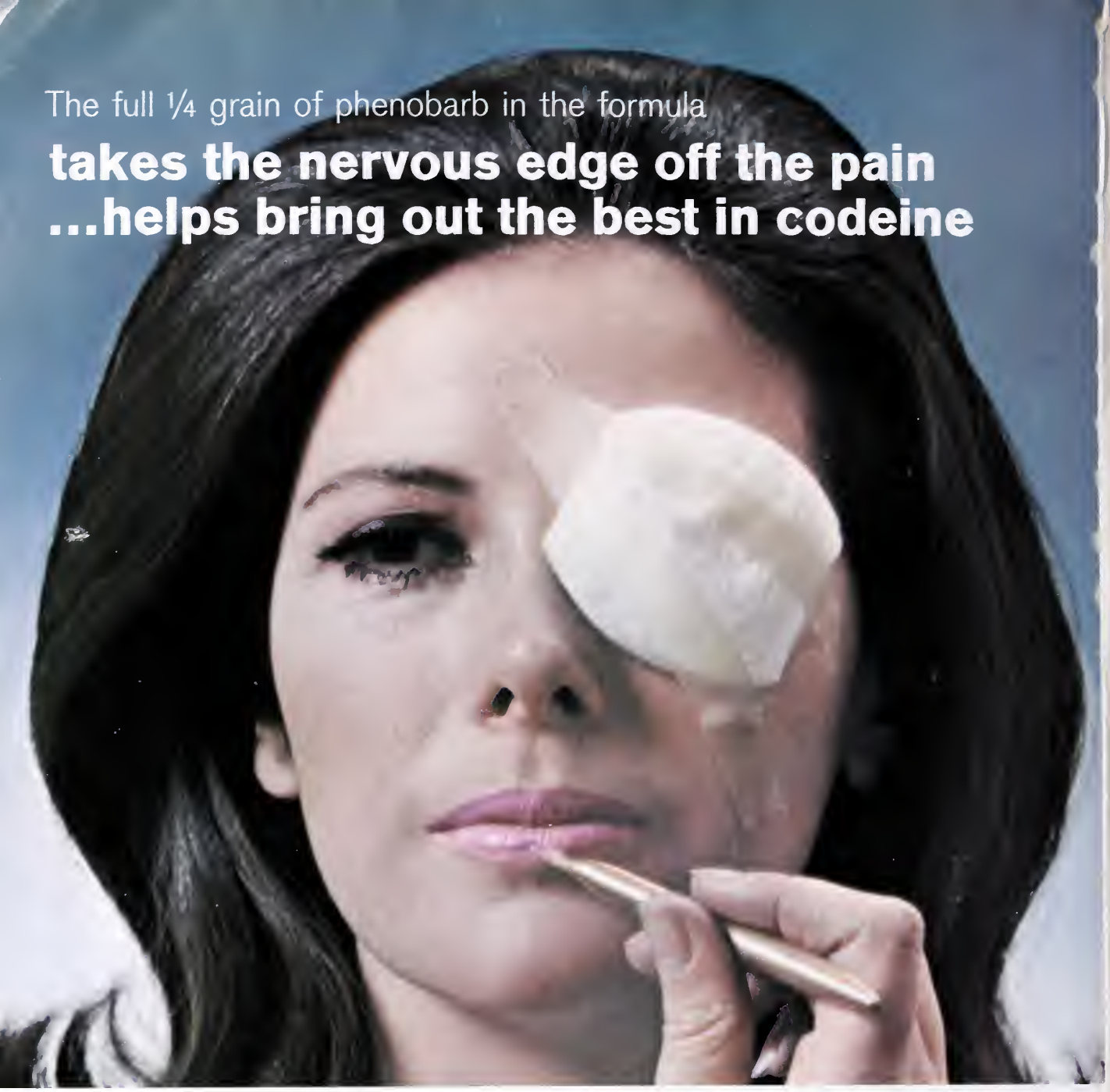

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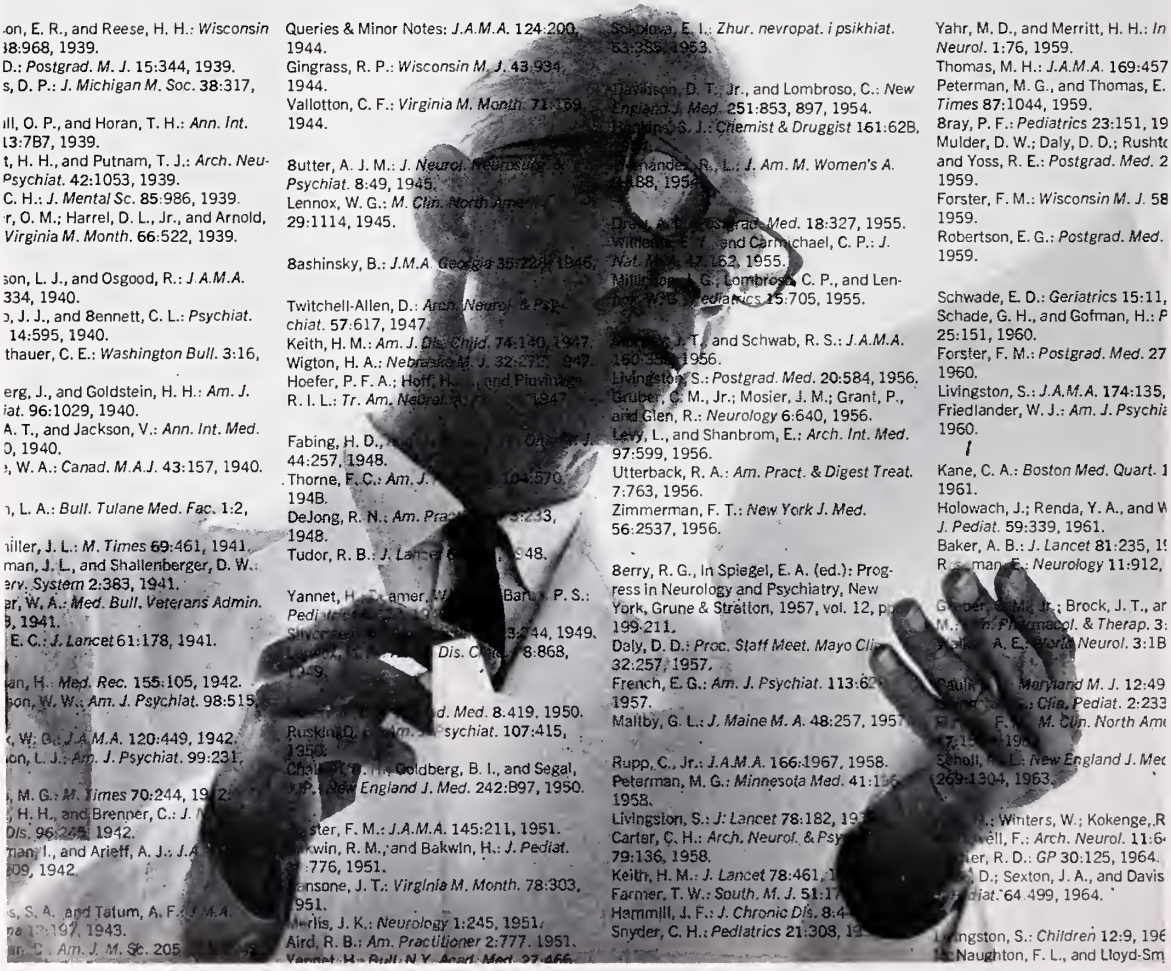

...on, E. R., and Reese, H. H.: *Wisconsin* ...8:968, 1939.
...D.: *Postgrad. M. J.* 15:344, 1939.
...s, D. P.: *J. Michigan M. Soc.* 38:317, ...
...ll, O. P., and Horan, T. H.: *Ann. Int.* ...13:7B7, 1939.
...t, H. H., and Putnam, T. J.: *Arch. Neu-* ...*Psychiat.* 42:1053, 1939.
...C. H.: *J. Mental Sc.* 85:986, 1939.
...r, O. M.; Harrel, D. L., Jr., and Arnold, ...*Virginia M. Month.* 66:522, 1939.

...son, L. J., and Osgood, R.: *J.A.M.A.* ...334, 1940.
...o, J. J., and Bennett, C. L.: *Psychiat.* ...14:595, 1940.
...thauer, C. E.: *Washington Bull.* 3:16, ...
...erg, J., and Goldstein, H. H.: *Am. J.* ...*iat.* 96:1029, 1940.
...A. T., and Jackson, V.: *Ann. Int. Med.* ...0, 1940.
...e, W. A.: *Canad. M.A.J.* 43:157, 1940.

...n, L. A.: *Bull. Tulane Med. Fac.* 1:2, ...
...iller, J. L.: *M. Times* 69:461, 1941.
...man, J. L., and Shallenberger, D. W.: ...*erv. System* 2:383, 1941.
...er, W. A.: *Med. Bull. Veterans Admin.* ...9, 1941.
... E. C.: *J. Lancet* 61:178, 1941.

...an, H.: *Med. Rec.* 155:105, 1942.
...son, W. W.: *Am. J. Psychiat.* 98:515, ...
...k, W. G.: *J.A.M.A.* 120:449, 1942.
...on, L. J.: *Am. J. Psychiat.* 99:231, ...
..., M. G.: *M. Times* 70:244, 1942.
..., H. H., and Brenner, C.: *J. N* ...*Dis.* 96:265, 1942.
...rian, I., and Arieff, A. J.: *J.A* ...109, 1942.

...s, S. A., and Tatum, A. F.: *J.A.M.A.* ...ne 121:197, 1943.
...n, C.: *Am. J. M. Sc.* 205 ...

Queries & Minor Notes: *J.A.M.A.* 124:200, 1944.
Gingrass, R. P.: *Wisconsin M. J.* 43:934, 1944.
Vallotton, C. F.: *Virginia M. Month.* 71:159, 1944.

Sutter, A. J. M.: *J. Neurol. Neurosurg. & Psychiat.* 8:49, 1945.
Lennox, W. G.: *M. Clin. North Amer.* 29:1114, 1945.

Sashinsky, B.: *J.M.A. Georgia* 35:228, 1946.

Twitchell-Allen, D.: *Arch. Neurol. & Psychiat.* 57:617, 1947.
Keith, H. M.: *Am. J. Dis. Child.* 74:140, 1947.
Wigton, H. A.: *Nebraska M. J.* 32:[illegible] 947.
Hoefer, P. F. A.; Hoff, H. ... and Pluvinage, R. I. L.: *Tr. Am. Neurol. A.* ... 1947.

Fabing, H. D., ... *Dis. Child.* 44:257, 1948.
Thorne, F. C.: *Am. J.* ... 104:570, 1948.
DeJong, R. N.: *Am. Pra...* 58:233, 1948.
Tudor, R. B.: *J. Lancet* ... 68:48.

Yannet, H. ... Kramer, W. ... Barr, P. S.: *Pediatrics* ... 3:744, 1949.
Silverman, ... *Dis. Child.* ... 8:868, ...9.

...d. Med. 8:419, 1950.
Ruskin, D. ... *Psychiat.* 107:415, 1950.
Chalfen, ... Goldberg, B. I., and Segal, ... *New England J. Med.* 242:897, 1950.

...ster, F. M.: *J.A.M.A.* 145:211, 1951.
...kwin, R. M., and Bakwin, H.: *J. Pediat.* ...:776, 1951.
...ansone, J. T.: *Virginia M. Month.* 78:303, 1951.
...erlis, J. K.: *Neurology* 1:245, 1951.
Aird, R. B.: *Am. Practitioner* 2:777, 1951.
Yannet, H.: *Bull. N.Y. Acad. Med.* 27:466, ...

Sokolova, E. I.: *Zhur. nevropat. i psikhiat.* 53:385, 1953.

Davidson, D. T., Jr., and Lombroso, C.: *New England J. Med.* 251:853, 897, 1954.
Hopkins, S. J.: *Chemist & Druggist* 161:628, ...
...hander, R., L.: *J. Am. M. Women's A.* ...:188, 1954.
Dodd, A. ... *Postgrad. Med.* 18:327, 1955.
Wilhelmi, E. T., and Carmichael, C. P.: *J. Nat. M. A.* 47:162, 1955.
Millichamp, G., Lombroso, C. P., and Lenox, W. G.: *Pediatrics* 15:705, 1955.
... T., and Schwab, R. S.: *J.A.M.A.* 160:35..., 1956.
Livingston, S.: *Postgrad. Med.* 20:584, 1956.
Gruber, C. M., Jr.; Mosier, J. M.; Grant, P., and Glen, R.: *Neurology* 6:640, 1956.
Levy, L., and Shanbrom, E.: *Arch. Int. Med.* 97:599, 1956.
Utterback, R. A.: *Am. Pract. & Digest Treat.* 7:763, 1956.
Zimmerman, F. T.: *New York J. Med.* 56:2537, 1956.

Berry, R. G., In Spiegel, E. A. (ed.): Progress in Neurology and Psychiatry, New York, Grune & Stratton, 1957, vol. 12, p... 199-211.
Daly, D. D.: *Proc. Staff Meet. Mayo Clin.* 32:257, 1957.
French, E. G.: *Am. J. Psychiat.* 113:6... 1957.
Maltby, G. L.: *J. Maine M. A.* 48:257, 1957.

Rupp, C., Jr.: *J.A.M.A.* 166:1967, 1958.
Peterman, M. G.: *Minnesota Med.* 41:1... 1958.
Livingston, S.: *J. Lancet* 78:182, 19...
Carter, C. H.: *Arch. Neurol. & Psy...* 79:136, 1958.
Keith, H. M.: *J. Lancet* 78:461, 1...
Farmer, T. W.: *South. M. J.* 51:17...
Hammill, J. F.: *J. Chronic Dis.* 8:4...
Snyder, C. H.: *Pediatrics* 21:308, 1...

Yahr, M. D., and Merritt, H. H.: *In Neurol.* 1:76, 1959.
Thomas, M. H.: *J.A.M.A.* 169:457
Peterman, M. G., and Thomas, E. *Times* 87:1044, 1959.
Bray, P. F.: *Pediatrics* 23:151, 19
Mulder, D. W.; Daly, D. D.; Rushto and Yoss, R. E.: *Postgrad. Med.* 2 1959.
Forster, F. M.: *Wisconsin M. J.* 58 1959.
Robertson, E. G.: *Postgrad. Med.* 1959.

Schwade, E. D.: *Geriatrics* 15:11,
Schade, G. H., and Gofman, H.: *P* 25:151, 1960.
Forster, F. M.: *Postgrad. Med.* 27 1960.
Livingston, S.: *J.A.M.A.* 174:135,
Friedlander, W. J.: *Am. J. Psychia* 1960.

Kane, C. A.: *Boston Med. Quart.* 1 1961.
Holowach, J.; Renda, Y. A., and W *J. Pediat.* 59:339, 1961.
Baker, A. B.: *J. Lancet* 81:235, 1
R... man, E.: *Neurology* 11:912, ...

...; Brock, J. T., ar
...*Pharmacol. & Therap.* 3:
... A. E.: *World Neurol.* 3:1B

...*Maryland M. J.* 12:49
... *Clin. Pediat.* 2:233
... M. *Clin. North Ame*
...*New England J. Med* 269:1304, 1963.

..., Winters, W.; Kokenge, R ...ell, F.: *Arch. Neurol.* 11:6
...ler, R. D.: *GP* 30:125, 1964.
... D.; Sexton, J. A., and Davis ...*diat.* 64:499, 1964.

...ingston, S.: *Children* 12:9, 196
... Naughton, F. L., and Lloyd-Sm

Symposium on Tropical Diseases

William Beaumont General Hospital, El Paso

(Editor's Note: This is the second of three parts of a Symposium on Tropical Diseases presented at William Beaumont General Hospital in El Paso. The third part will be published in the October issue of SOUTHWESTERN MEDICINE.)

SCRUB TYPHUS

Maj. Ralph F. Wells, MC*

Scrub typhus or mite-borne typhus is an acute febrile disease caused by the rickettsiae, *R. tsutsugamushi* or *R. orientalis*. Clinically, it is typhus-like with a generalized rash, an eschar at the site of mite attachment, and regional or generalized lymphadenopathy. The prodromata are vague; the onset usually abrupt. The acute febrile phase is ordinarily of two weeks' duration, and the convalescent phase, if untreated, is prolonged. Agglutinins for Proteus OX-K usually may be demonstrated by the end of the second week though definitive diagnosis depends on isolation of the rickettsia from the patient's blood. This disease has a number of synonyms including Tsutsugamushi disease, Shishito disease, Kedani fever, rural typhus, tropical typhus and Japanese river or flood fever.

The geographic distribution of this disease is a roughly triangular area extending from Japan to India to Australia and back to Japan. Although demonstrating a seasonal pattern in Japan, the disease may be encountered year round in Viet Nam, Laos, Cambodia, Thailand, and Malaysia. The distribution in the latter areas tends to be patchy and is dependent on both the vegetation and native agricultural methods. "Krai" agriculture, which is basically a policy of cut, burn and abandon, sets the stage for "scrub growth."[4] This provides an ideal environment for the vector mites *Trombicula akamushi* or *Trombicula deliensis*. In addition, the grass *Imperate cylindrica* known colloquially as "kunai" or "lallang" grass provides good cover for rodents and the specific vector mites. From a military standpoint abandoned native villages or farm lands, new beachheads, or newly occupied territory in endemic areas pose a particular threat.[2,3,5]

The military significance of this disease cannot be over emphasized. The Japanese had been aware of the clinical picture in Japan for over 150 years. They had been aware of the vector and appropriate environmental control measures for nearly 75 years, yet in World War II they failed to recognize the Southeast Asia variant of the disease and sustained in excess of 30,000 casualties. Among Allied Forces in the China-Burma-India Theater and in the Southwest Pacific approximately 18,000 cases were reported. In 11,000 cases for which adequate

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data is available, there were about 650 deaths. For American troops death rates varied between 0.6 and 35.3 per cent in different epidemics. Other series report up to 60 per cent mortality. I do not have data on incidence of this disease in Viet Nam during the present conflict. Personal correspondence indicates that isolated cases have been recognized.[6]

The causative agent, *R. tsutsugamushi,* has recently been studied by electron microscopy revealing an outer envelope, an inner matrix and dense cytoplasmic granules typical of a rickettsial pattern. It is transmitted to man by the six legged larval stage of the trombiculid mites mentioned earlier. Other vectors, such as *T. scutellaris,* account for sporadic outbreaks. In the mite the rickettsiae are passed by the transovarian route. The six legged larva which transmits the disease to man feeds only once. Field mice, rats, voles, shrews, and other small animals harbor the larva and transmit the organism to new hosts. There is an alternate mite-rodent-mite cycle.

The clinical picture, diagnosis, management and prevention of this disease are of chief interest to us as physicians. The basic pathology is a vasculitis capable of affecting skin, myocardium, lungs and the central nervous system. Readers are referred to standard texts for a more elaborate discussion of the pathology.

After an incubation period of 6-21 days (usually 10-12) the patient experiences abrupt onset of chills, fever, conjunctival injection and retro-bulbar headache. The fever increases stepwise to 102° to 105° by the end of the first week, remains elevated the second week, and falls by lysis at the beginning of the third week. The modification induced in this pattern by antibiotics will be discussed shortly.

Between the fifth and tenth day a characteristic red, macular rash may be seen, but the key cutaneous feature is the eschar. This occurs in most Caucasians and in 10-20 per cent of Asians. It appears on the fourth or fifth day at the site of mite attachment as a papule 8 or 10 mm in diameter and 2 mm in height. This goes on to ulcerate and later becomes encrusted. The original bite is painless, and there is a predilection for areas where clothes bind or where opposing cutaneous surfaces are in contact as in the axilla or perineum. The eschar epithelializes after defervescence. The rash mentioned above is often evanescent, fading, and reappearing. It usually is seen on the trunk but may also erupt on the extremities or on the palate.

Regional adenopathy occurs in relation to the eschar; generalized lymphadenopathy and splenomegaly occur, a reflection of the generalized rickettsemia. In addition, splenomegaly may be related to coexistant malaria. The spleen is usually detected at the end of the first week.

A spectrum of central nervous system symptoms appears ranging from delirium to coma, but inevitably accompanied by intense retro-bulbar or retro-ocular headache. Deafness which spontaneously improves has been noted frequently.

Variable degrees of myocarditis have been observed. There may be severe tachycardia, hypotension, and congestive heart failure or conversely, a bradycardia disproportionate to the high temperature. Respiratory symptoms and pulmonary findings are less dramatic, though pneumonitis may occur in some patients.

Convalescence in the past was rather protracted; it has been speeded considerably by the advent of antibiotic therapy. Resistance to homologous strain infection may last several years, though a second attack with a heterologous strain may occur within months. Prognosis is affected by age, morbidity and mortality; it is poorer in children and patients over 40. The outlook is also affected by the presence of other diseases such as malaria, hepatitis, or dysentery.

The diagnosis is suggested by the pattern of leukopenia during the first week with a subsequent white blood cell increase to 12,000 or 15,000/mm.^{3} Most useful of the screening studies is the presence of agglutinins to Proteus OX-K. A titre of 1:160 is highly suggestive but more important is either a rise or fall in titre. A negative test does not, however, exclude scrub typhus nor does a specific positive test establish the diagnosis; non-specific rises in titre of OX-K may be evoked by other infections such as relapsing fever. Complement fixation techniques have not proved reliable.

Isolation of rickettsia from the patient's blood

confirms the diagnosis. Rickettsemia persists 8-10 days in the untreated patient and lasts for at least 24 hours in the patient receiving antibiotics. After appropriate preparation the suspect blood may be injected intraperitoneally into mice; this usually proves fatal in 10-16 days. Giemsa stained smears of peritoneal scrapings or splenic impressions may demonstrate rickettsiae.

Chloramphenicol and the tetracyclines are specific therapeutic agents against scrub typhus. These antibiotics are apparently rickettsiostatic; however, fever and symptoms usually respond within 24-48 hours. A recommended regimen for chloramphenicol is 3.0 grams as a loading dose followed by 0.5 grams q 6 h until the temperature is normal. Since the drug is only rickettsiostatic and since immunity does not develop until the 14th day, a relapse may occur a week or so after completion of treatment. Although the relapse responds to the same agent it may be prevented by administering a 3.0 gram oral dose of chloramphenicol six days after completion of the first course.[3]

No effective immunization has been developed. Readers are referred to TB Med 31[1] for a discussion of individual preventive measures. In the rare situation where men are assigned to a high risk area and individual protective measures will be impractical, prophylactic antibiotics might be advisable, although under no circumstances should they be administered in excess of six weeks.

REFERENCES

1. TB Med 31: Scrub Typhus (Mite-borne typhus fever, Tsutsugamushi disease) 1962.
2. Hunter, G. W., Frey, W. W., and Swartzwelder, J. C.: A Manual of Tropical Medicine, 3d Edition, Philadelphia, W. B. Saunders, 1960, p 94-101.
3. Control of Communicable Disease in Man, 10th Edition, New York, The American Public Health Association, 1965 p. 211-213.
4. Tipton, V. J.: Personal Communication, 1965.
5. Arthropod-Borne Rickettsial Diseases, Medical Field Service School, Department of Preventive Medicine, LP 72-198-101, 035. 1965.
6. Blount, R. E.: Personal Communication. 1966.

TROPICAL SPRUE

A recent paper [1] reporting a 12 per cent incidence of tropical sprue among a selected group of Special Forces completing six months in Southeast Asia has again focused the military physician's interest on this very intriguing disease. Although described by Hillary[2] in 1759, the military significance of this disease was not fully appreciated until World War II when for the first time the Western Powers had sizeable manpower commitments in Southeast Asia. Ironically, one of the finest studies was conducted by Steffanini[3] in an Italian P.O.W. camp located in the Yol-Kangra Valley in the foothills of the Himalayas. He studied 1069 cases between September 1942 and the end of April 1945. This represented an incidence of about 3.5 per cent of the camp population. Keele and Bound[4] reported that among 8,846 British troops medically evacuated from India, 1,073 suffered from sprue. In several RAF units morbidity rates varied from 10-50 per cent in the first few months following arrival in India. Although precise figures are not available, the United States' experience in the China-Burma-India Theater proved sufficient impetus to establish the United States Army Tropical Research Medical Laboratory in San Juan, Puerto Rico. One of the major contributions of this unit has been development of the Crosby small bowel biopsy capsule[5,6]. The unit is presently being phased out but had studied over 125 cases of tropical sprue at the time its clinical activities were suspended.

The purpose of this paper is to review the clinical picture of tropical sprue and to review the differences between this disease and non-tropical sprue (gluten induced enteropathy). The scope of this paper does not allow a detailed discussion of malabsorption. The reader is referred to several excellent reviews on this subject[7,8,9,10,21].

Tropical sprue has been defined[11] as a disease of unknown etiology characterized by varying degrees of villous atrophy of the small intestine, malabsorption and the subsequent development of multiple nutritional deficiencies. Malabsorption of folic acid and vitamin B_{12} commonly results in the development of megaloblastic anemia. It is endemic in large areas of the Far East including most of India and Southeast Asia.

In the Caribbean, tropical sprue has been reported in Puerto Rico and the Barbados but curiously has spared Jamaica as it has the African continent. In addition to sporadic cases, large scale epidemics of sprue have been reported from India.

An epidemic in 1960 and 1961 covered 5,000 square miles, afflicted 100,000 people and had a mortality rate of 10 per cent. While return to a temperate climate usually results in spontaneous improvement, tropical sprue has first appeared or has undergone transition from an acute to a chronic state in these circumstances.

Much difficulty has been encountered in forming a working concept of tropical sprue. This may be true because different observers have studied this disorder at various degrees of severity. The majority of the patients described in the Caribbean have macrocytic anemia and a megaloblastic bone marrow. In contrast, British workers in the Far East have regarded gastrointestinal manifestations as predominant and have found anemia in only 20 per cent of their patients.

Steffanini proposed a practical classification of the clinical stages of tropical sprue which has been modified recently by Gardner.[12] The early phase (first stage) is characterized by fatigue and asthenia. Bulky stools rather than diarrhea may be the initial bowel disorder. The severe asthenia is related to electrolyte depletion, especially sodium and potassium ions. Small bowel function may be abnormal and if measured, steatorrhea is present. American military personnel in Puerto Rico predominantly were in this phase.

After an interval of a few weeks to months, a deficiency phase (second stage) develops. Glossitis, stomatitis, cheilosis, and hyperkeratosis are more prominent. Weight loss becomes more prominent, reflecting dysphagia and malabsorption. Moderate reductions in prothrombin may occur; hypocalcemia and tetany are rare. All laboratory measurements of absorption are impaired and more pronounced than in the early phase. The majority of British military patients studied in India were in this category.

With progressive malabsorption the bone marrow becomes megaloblastic. This represents the macrocytic anemia phase (third stage). Weight loss is severe; the musculature wasted. Diarrhea and abdominal distension may not be prominent as the patient's food intake decreases due to lassitude and anorexia. Fourteen per cent of Steffanini's patients were in this category, and it is also frequently seen in Cuba and Puerto Rico.

As indicated earlier, rather than comprehensively reviewing the various laboratory studies available for assessing malabsorption I should like to discuss only three: small bowel x-ray, intestinal biopsy, and the hematologic picture.

Segmentation and flocculation of the barium and dilatation of the small bowel is similar if not identical with the appearance in non-tropical sprue. Early in the course the x-ray may be normal. There is good correlation between the small bowel biopsy and roentgen changes. Conversely, although gastric mucosal changes are striking both functionally and on biopsy, there is no characteristic x-ray abnormality of the stomach.[13, 14] With remission, the small bowel pattern improves. The mechanism of the small bowel abnormality is unknown.

The role of small bowel biopsy in the study of malabsorption has recently been assessed as has the status of electron microscopy.[15, 16, 17] Sheehy[18] has classified the results of his jejunal biopsies into three categories: subtotal villous atrophy, partial villous atrophy, and normal. Subtotal villous atrophy represents the most serious involvement with a flat mucosal surface devoid of villi. The epithelium was cuboidal, the crypts reduced in number but elongated, and the number of goblet cells usually increased.

Partial villous atrophy represents a less severe degree of involvement but is characterized by abnormal villi which are usually short, broad, and squat or clubbed, ranging from 100-250 micra in height. Normal mucosa showed long fingerlike villi with a well preserved brush border. The presence of infiltration in the lamina propria was also noted and found to correlate well with the degree of functional and radiographic derangement. Improvement in the microscopic appearance of the jejunal mucosa was found in biopsies from 15 of 20 patients after two to three years of folic acid therapy. Seven patients appeared to have normal villi at the conclusion of the study. The infiltration in the lamina propria had disappeared in seven and decreased in four of a group of 11 patients. A second group of 26 patients with chronic tropical sprue fall into the category of subtotal villous

atrophy. A slightly different classification accompanied by superb illustrations is presented by Swanson and Thomassen.[22]

The hematologic features of this disease are among the most unique and help to distinguish it from non-tropical sprue. In the early and deficiency stages, anemia is not a major problem. Military patients in Puerto Rico were found to have normal serum iron and no anemia. In contrast, when patients had megaloblastic bone marrow alterations, serum irons were depressed. Attempts to perform an iron tolerance test were unsatisfactory. Many of these patients in Stage III will have a dimorphic anemia with iron deficiency and a megaloblastic anemia indistinguishable morphologically from Addisonian pernicious anemia. The macrocytic red cells when tagged with Cr^{51} have been found to have a half time of 12 as opposed to a normal of 28 days. During hematologic remission the life span returns to normal. Tagged red cells from patients with untreated sprue when transfused into normal recipients, had a life span of 12 days, again much similar to pernicious anemia. Steffanini reported five patients who progressed to aplastic anemia.

Steffanini's[3] work with dietary measures and the administration of crude liver extract represented the first breakthrough in the therapy of tropical sprue other than evacuation of the endemic area. As more knowledge has accrued it has been found that folic acid in a dose of 15 milligrams per day will produce a symptomatic remission, and a gradual histologic and hematologic response. Vitamin B_{12} has proved of value in the correction of the anemia. The broad spectrum antibiotics including the tetracyclines and chloramphenicol have induced a hematologic remission and reduced steatorrhea in one-third of the patients in whom they have been tried.[19] Their mechanism of action is obscure as the normal bowel flora are only now being accurately identified.[20] There is no significant response to a gluten free diet in this disease.

Summary

The last 20 years have seen the histopathologic basis of tropical sprue identified, the basic pathophysiology better delineated, and the evolution of a rational, effective therapeutic program. Despite these advances, the basic etiology remains obscure. Current theories about etiology which warrant further investigation are infection, dietary deficiency, and the role of "rancid" fats in the succus entericus. Tropical sprue, unlike non-tropical sprue, does not respond to a gluten free diet. A favorable therapeutic response may be obtained with folic acid, Vitamin B_{12} and the broad spectrum antibiotics. The diseases may also be distinguished by the locale in which they occur, and, finally, some distinction may be made on a histologic basis.

References

1. Sheehy, T. W., Cohen, W. D., Wallace, D. K., and Legers, L. L.: Tropical Sprue in North Americans, JAMA 194: 1069 (1965).
2. Both, C. C.: The First Description of Tropical Sprue, Gut 5:45, (1964).
3. Steffanini, M.: Clinical Features and Pathogenesis of Tropical Sprue, Medicine 27:379 (1948).
4. Keele, K. D. and Bound, J. P.: Sprue in India, Best Med J 1:77 (1946).
5. Crosby, W. M. and Kugler, H. W.: Intraluminal Biopsy of the Small Intestine: The Intestinal Biopsy Capsule, Am. J. Dig. Dis. 2:236 (1957).
6. Sheehy, T. W.: Intestinal Biopsy, Lancet 1:959 (1964).
7. Laster, L. and Ingelfinger, F. J.: Intestinal Absorption-Aspects of Structure, Function and Disease of the Small Intestine, New Eng. J. Med. 261:1138, 1192, 1246 (1961).
8. Benson, G. D., Kowlesar, D. D., and Sliesenger, M. H.: Adult Celiac Disease with Emphasis upon Response to the Gluten-Free Diet, Medicine, 43:1, (1964).
9. Badenoch, J.: Steatorrhea in the Adult, Brit Med J. 2:879, 963 (1960).
10. Jeffries, G. H., Weser, E., and Sleisenger, M. H.: Malabsorption, Gastroenterology 46:434 (1964).
11. Klipstein, F. A.: Tropical Sprue in New York City, Gastroenterology 47:457 (1964).
12. Gardner, F. M.: Tropical Sprue, New Eng. J. Med. 258: 791, 835, (1958).
13. Vaish, S. K., Sampathkumas, J., Jacob, R. and Baker, S. J.: The Stomach in Tropical Sprue, Gut 6:458. 1965.
14. Flock, M. H., Thomassen, R. W., Cox, R. S. and Sheehy, T. W.: The Gastric Mucosa in Tropical Sprue. Gastroenterology 44:567 (1963).
15. Shiver, M.: Duodenal Biopsy, Lancet 1:17 (1956).
16. Ruben, E. E. and Dobbins, W. O. III,: Peroral Biopsy of the Small Intestine. A Review of its Diagnostic Usefulness. Gastroenterology 49:676 (1965).
17. Trier, J. S. and Ruben, C. E.: Electron Microscopy of the Small Intestine: A Review. Gastroenterology 49:574 (1965).
18. Sheehy, T. W., Baggs, B., Perez-Santiago, E. and Flock, M. H.: Prognosis of Tropical Sprue. Ann Int. Med 57:892 (1962).
19. Sheehy, T. W. and Perez-Santiago, E.: Antibiotic Therapy in Tropical Sprue, Gastroenterology 41:208 (1961).
20. Shiner, M., Waters, T. E. and Gray, J. D. A.: Culture Studies of the Gastrointestinal Tract with a Newly Devised Capsule, Gastroenterology 45:625 (1963).
21. Conference on Intestinal Malabsorption and Allied Hematologic Problems. Amer. J. Di. Dis. 9:769, (1964).
22. Swanson, V. L. and Thomassen, R. W.: Pathology of the Jejunal Mucosa in Tropical Sprue. Amer. J. Clin. Path. 46:511 (1965).
23. Guerra, R., Wheby, M. S. and Bayless, T. M.: Long-term Antibiotic Therapy in Tropical Sprue, Ann. Int. Med. 63: 619 (1965).

Some Difficult Obstetrical Cases

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A mother of nine in her thirties developed hypertension, albuminuria and edema in the last trimester. She was a forceful, good person—a community leader. We re-checked her every two or three days at the office, treating her "conservatively" with sedation, rest, low salt, diuresis and anti-vasospastic drugs. She was seized one night by severe lumbar pain. In the hospital nystagmus was noted, and she began convulsing. We ruptured the membranes and controlled the convulsions with sodium amytal intravenously. She went into labor. The cervix was ripe. Nine hours later she and the baby were dead. In the years that followed I observed her family living without her—and a community without an active leader.

When she presented with hypertension, albuminuria and edema, we should have ruptured the membranes THEN, and had we done so, she would have been alive today. It was once "conservative" in abortion NOT to curette—"what nature started, nature will end"—but now we have learned the fallacy of that. Sedation, diuretics, rest, anti-vasospastics do not resolve the pathological process that is threatening the life and health of a mother and the fetus, but on the contrary these methods mask the manifestations of the process and lull us into a false sense of security. Termination of pregnancy does reverse the pathological process.

A young, vigorous primipara with an over-distended uterus presented herself at the hospital with a small foot protruding from the vagina. The size of the foot suggested a multiple pregnancy. Being "conservative" by training and instinct, I took her to the delivery room and made gentle but firm traction on the foot. She was given 100 per cent oxygen by re-breathing orofacial mask and saddle-block anesthesia by 5 mgm. of Pontocaine* in Dextrose 5 per cent in Lumbar IV, 1.6 cc. She had vigorous contractions. She was at or near term, the cervix was "ripe" and soon dilated or tore enough for a skillful breech delivery with Piper forceps on the after-coming head. Immediately I inserted my hand into the uterus, ruptured the amnion of the second twin, and did a skillful version-extraction ended with Piper forceps. Both babies were pale, flaccid with good pulse but responded promptly to oxygen under positive pressure. Then the mother began to bleed fiercely. Repeated ergonovine, pitocin, massage and bimanual compression failed to staunch the bleeding. Transfusions were ordered, and four units were given in sequence. Unable to see into the vagina with available lighting, I packed the vagina tightly with gauze—I was taught never to pack a bleeding uterus. Finally the bleeding stopped, and the mother and twin boys made a healthful recovery.

In retrospect, experts agree that a country doctor was in deep trouble, that the footling should have been bathed with pHisohex, and that a cesarean section should have been done. A valuable technique for the control of postpartum hemorrhage that does not appear in the text-books is as follows: (1) An ordinary obstetrical forceps blade makes a most efficient retractor for the postpartum vagina, using the blade of the opposite side with concavity outward applied against the lateral wall of the vagina and held by both hands of an assistant either with sterile gloves or through the sterile drapes. (2) A good operating light capable of casting a beam deep into the vagina is necessary. (3) Four curved, serrated sponge forceps are available, and one is placed on the anterior lip of the cervix and another on the posterior lip, drawing the uterus down to the introitus and retracting it laterally away from the forceps blade to expose the lateral fornix of the vagina. (4) The third curved, serrated sponge forceps is placed in the lateral fornix of the vagina grasping the thinned out postpartum lower uterine segment at about the point of entry of the uterine artery—mindful of the position of the bladder anteriorly—closing the sponge forcep with one "click". (5)

*Winthrop Laboratories

If the bleeding has not stopped, the procedure is repeated on the opposite side, using the other obstetrical forceps blade and the fourth curved, serrated sponge forceps. The sponge forceps may be left in place for two hours. They are quite painful. If this procedure does not control bleeding, it is probable that hysterectomy will be required.

A 45-year-old mother of seven in her 11th pregnancy developed hypertension, albuminuria and edema in the eighth month of pregnancy. The cervix felt "ripe" and the vertex presented: so without ado we ruptured the membranes. The next day labor was established but not vigorous, and after eight hours the cervix was still only two fingers dilated, and meconium told us that our "vertex" was a frank breech. This was confirmed by X-Ray. The fetal heart was good: so we permitted four more hours of active labor, and by this time I was able to make traction by my finger in the posterior fetal groin working with contractions to decompose the breech and wipe down the arms, but the head was tightly stuck in the lower uterine segment. A Mariceau maneuver was attempted but failed: so Piper forceps were applied to the after-coming head, promptly delivering the fetus. But the Apgar was zero. There was a moderate postpartum hemorrhage not requiring transfusion. The mother promptly recovered from the toxemia.

In retrospect, we should have done a cesarean section on this 45-year-old mother with a frank breech. Perhaps we were taught to be too "conservative" about cesarean section in the pre-antibiotic era of obstetrics. At section we could also have occluded the tubes to prevent another unwise pregnancy.

401 N. 4th St.

NEW OFFICERS—Dr. Paul A. Feil, Deming, center, was elected President of the New Mexico Chapter of the American Academy of General Practice at the Ruidoso Summer Clinic in Ruidoso, N.M., July 25-30, 1966. Other officers are, left to right, Dr. James A. Koch, Albuquerque, retiring President, Dr. C. W. Carroll, Las Cruces, Secretary-Treasurer, Dr. Edward D. Fikany, Fort Sumner, President-Elect, Dr. Henry L. Wall, Artesia, Vice-President, Dr. Hubert R. Teague, Albuquerque, Director, and Dr. Jose A. Rivas, Belen, Delegate to the AAGP. Not shown are Dr. Clarence H. Peterson, Carlsbad, new Director, and Dr. U. S. Marshall, Roswell, AAGP Delegate.

Hospital Coronary Care Unit Is First of Kind in Houston

Houston's first exclusive facility for coronary intensive care officially opened August 16 at St. Joseph Hospital.

The six-bed unit is equipped with $55,415 worth of the most advanced electronic monitoring equipment. This equipment was purchased with funds made available by a $500,000 gift to the St. Joseph Hospital Foundation from Mr. and Mrs. George W. Strake.

In order to staff the unit 24 hours a day, 21 Hospital Staff members have already completed a 54-hour course in intensive care of coronary patients.

The unit was designed according to specifications outlined by the United States Public Health Service, and thus qualifies for a $60,000 one-year operating, cost-sharing contract. Holy Cross Hospital in Silver Springs, Maryland, is the only other community hospital to receive a similar contract from the Public Health Service.

The Director of the Coronary Care Unit is assisted in his duties by two Staff Physicians.

Specially-trained nursing personnel, staffing the unit on a 24-hour-a-day basis, include a team of three for each eight-hour shift. In addition, the unit also has a secretary and a ward clerk. All beds in the unit can be observed from the central nursing station and each patient is continuously monitored by electronic equipment. Should any irregularities occur, the nurses are trained to recognize them, and to administer emergency treatment until a physician arrives. Specially-trained nurses are considered an essential part of the success of a coronary intensive care unit.

The member of the House Staff on call sleeps in quarters adjacent to the unit. An Anesthesiologist is available for emergencies 24 hours daily, also sleeping nearby.

Private physicians retain their roles as attending physicians in the same manner as they would on regular nursing floors. Cardiovascular consultations may be requested if desired. Except in emergency situations, supervisory medical personnel do not converse with patients, make examinations or question the attending physician's orders.

Future plans include formulation of a teaching program to teach Coronary Intensive Care to senior students at Sacred Heart Dominican College, Department of Nursing; representatives of other hospitals operated by the Sisters of Charity; and finally members of the staffs of other hospitals.

Southwestern Medicine Now Available on Microfilm

SOUTHWESTERN MEDICINE has entered into an agreement with University Microfilms, Inc., Ann Arbor, Michigan, to make available to libraries issues of SOUTHWESTERN MEDICINE in microfilm form.

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First Fall AAGP Assembly to be Held in Boston

The first fall Scientific Assembly of the American Academy of General Practice will be held October 10-13, 1966, in Boston.

Among subjects to be discussed are adolescent problems, stroke, obstetrics and mental retardation, and heart disease. Two half-days of bedside refresher courses at Massachusetts General and 13 other hospitals are scheduled.

The four-day postgraduate educational meeting, which annually draws some 3,000 family physicians from throughout the United States, will present 31 medical authorities and some 115 scientific exhibits keyed to the program. The scientific program will follow the annual meeting of the Academy's Congress of Delegates, October 8-10, in the Sheraton Boston Hotel.

Among speakers will be Dr. Maynard I. Shapiro, Chairman of the Academy's Commission on Education, Judge Leo B. Blessing, New Orleans, Judge of the Orleans Parish Juvenile Court, Dr. J. Roswell Gallagher, Chief of the Adolescents' Unit at Boston Children's Hospital and Clinical Professor of Pediatrics at Harvard Medical School, Dr. Edward J. Kowalewski, Akron, Pennsylvania, Academy Vice-President Dr. Francis L. Land, Dr. S. J. Behrman, Professor of Obstetrics and Gynecology at the University of Michigan, Tuft's Dr. Edward F. Rabe and Harvard's Dr. Cesare T. Lombroso.

Clinical refresher courses will be conducted by Faculty members from Harvard, Tufts and Boston University Medical Schools, with 14 Boston hospitals taking part in the program. There will be nearly 60 courses offered each day on topics ranging from treatment of cardiac arrest to leukemia and office gynecology. The courses will be conducted for groups of five to 10 participants.

This year's Assembly will feature two floors of scientific exhibits and more than 30 lectures in the new War Memorial Auditorium. On Tuesday morning, "Therapeutic Nuggets," will offer physicians five 10-minute capsulizations of latest developments in therapy and procedures, ranging from nitrogen mustard injections for arthritis to mediastinoscopy.

Other topics will be "Diabetic Neuropathies," by Dr. Priscilla White of Boston's famed Joslin Clinic; a symposium on cardiac arrest and the current status of cardiac pacemakers; food contamination; infant orthopaedic problems and hematologic problems, which will be covered by closed circuit color television, using live patients, being transmitted into the lecture hall.

Coming Meetings

Flying Physicians Association, Las Vegas, Nevada, The Dunes Hotel and Country Club, September 11-16, 1966.

96th Annual Session of The Colorado Medical Society, Colorado Springs, The Broadmoor, September 25-28, 1966.

Texas Dermatological Society, El Paso, October 1-2, 1966.

Western Orthopedic Association, Tucson, Ramada Inn, October 2-6, 1966.

American Society of Plastic and Reconstructive Surgeons, Las Vegas, Nevada, Flamingo Hotel, October 2-7, 1966.

Medical Society of the United States and Mexico, Tucson, Pioneer Hotel, October 5-7, 1966.

American College of Surgeons, San Francisco, Fairmont Hotel, October 10-14, 1966.

American College of Obstetricians and Gynecologists, Houston, October 24-26, 1966.

American Association of Public Health Physicians, San Francisco, October 31-November 4, 1966.

Interim Meeting of the New Mexico Medical Society, Alamogordo, New Mexico, Holiday Inn, November 11-12, 1966.

Western Surgical Association, Phoenix, Del Webb's Townehouse, November 16-19, 1966.

Eight National Conference on the Medical Aspects of Sports, in conjunction with the 20th Annual Clinical Convention of the AMA, Las Vegas, Nevada, Caesar's Palace, November 27-30, 1966.

Southwest Allergy Forum, Galveston, Flagship Hotel, January 19-21, 1967.

American College of Surgeons, Arizona Chapter, Tucson, Arizona Inn, January 20-21, 1967.

TMA District 1 Meeting, Pecos, February 4, 1967.

American Academy of Pediatrics, San Francisco, Hilton Hotel, April 3-4, 1967.

American Society of Internal Medicine, San Francisco, St. Francis Hotel, April 7-9, 1967.

Southwestern Surgical Congress, Phoenix, Del Webb's Townehouse, April 10-13, 1967.

American College of Physicians, San Francisco, Fairmont Hotel, April 10-14, 1967.

American Academy of Neurology, San Francisco, Hilton Hotel, April 24-29, 1967.

Arizona Medical Association, Phoenix, Townehouse, April 26-29, 1967.

American Cancer Society, Dallas, Sheraton-Dallas Hotel, May 3, 1967.

American Gynecological Society, Phoenix, Arizona Biltmore Hotel, May 4-6, 1967.

Texas Medical Association, Dallas, May 4-7, 1967.

85th Annual Meeting of the New Mexico Medical Society, Santa Fe, La Fonda Hotel, May 18-20, 1967.

48th Biennial Meeting of the Southwestern Medical Association, El Paso, Sheraton-El Paso Motor Inn, February 8-10, 1968.

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Official Journal of the Southwestern Medical Association,
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Texas District One Medical Association, The Southwestern New Mexico Medical Society,
and El Paso County Medical Society

IN THIS ISSUE

VOL. 47, NO. 10

October, 1966

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Official Journal of

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VOL. 47 OCTOBER NO. 10

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*Schiller, I. W., and Lowell, F. C.: New England J. Med. 261:478, 1959.

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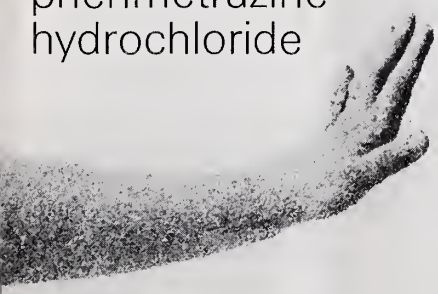

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N. M. Medical Society
Interim Meet, Nov. 10-12

Chemotherapy and Hypertension will be discussed at the Interim Meeting of the New Mexico Medical Society November 10-12, 1966, Holiday Inn, Alamogordo, New Mexico.

Speakers on the scientific agenda will be Dr. Grant Taylor, Houston, Chairman of the Southwest Chemotherapy Study Group, Dr. Laurens P. White, San Francisco, Dr. Robert K. Seller, Philadelphia, Assistant Professor of Medicine, Hahnemann Medical College and Hospital, Dr. Ray W. Gifford, Jr., Cleveland Clinic, Dr. Robert Whang, Albuquerque, Assistant Professor of Medicine, University of New Mexico School of Medicine.

The Council will meet at 2 p.m., Thursday, November 10, and the House of Delegates will meet at 2 p.m. on Friday and Saturday. A banquet, with speaker to be announced, will be held at 7:30 p.m. Friday.

Dr. William La Barre, Alamogordo, President of the Otero County Medical Society, will welcome physicians. Dr. Melvin A. Lester, Alamogordo, is chairman of arrangements for the meeting and is being assisted by Dr. Richard C. Sherman and Dr. Margaret Janson, both of Alamogordo. Registration fee is $10. Mead Johnson Laboratories is sponsoring the Chemotherapy of Cancer scientific session and Merck Sharp & Dohme is sponsoring the Hypertension session.

The complete program is as follows:

Thursday, November 10

1:00 p.m. Registration
Fee $10.00.

2:00 p.m. Council Meeting

6:30 p.m. Dinner for Councilors at Holiday Inn

Friday, November 11
Chemotherapy of Cancer Session
Sponsored by Mead Johnson Laboratories

7:30 a.m. Registration

8:30 a.m. Welcome and Introductory Remarks — William La Barre, M.D., President, Otero County Medical Society

8:45-9:30 a.m. General Discussion of Cancer Chemotherapy
Grant Taylor, M.D., Chairman,
Southwest Cancer Chemotherapy Study Group,
Houston, Texas

9:30-10:15 a.m. Chemotherapy of Lymphomas
Laurens P. White, M.D., Internal Medicine, San Francisco, California.

10:15-10:30 a.m. Coffee Break

10:30-11:15 a.m. Cancer Chemotherapy
Topic of Dr. Taylor's Choice

11:15-12:00 Noon. Chemotherapy of Melanoma
Laurens P. White, M.D.

12:00-12:30 p.m. Question and Answer Period
Drs. White and Taylor

12:30-2:00 p.m. Lunch Break

2:00 p.m. House of Delegates Meeting
(First Session)

3:30 p.m. Reference Committee Hearings

6:30 p.m. Cocktails (Cash Bar)

7:30 p.m. Banquet (purchase tickets at registration desk)
Guest Speaker — to be announced

Saturday, November 12
Hypertension Session

Sponsored by Merck Sharp & Dohme

8:30 a.m. Introduction of Speakers

8:45-9:30 a.m. Diagnostic Workup of Patients with Hypertension
Robert K. Seller, M.D.,
Assistant Professor of Medicine
Hahnemann Medical College & Hospital,
Philadelphia, Pa.

9:30-10:15 a.m. Management of Essential Hypertension & Hypertensive Crises
Ray W. Gifford Jr., M.D.,
Cleveland Clinic, Cleveland, Ohio

10:15-10:30 a.m. Coffee Break

10:30-11:15 a.m. Management of Renal Hypertension
Robert Whang, M.D.,
Assistant Professor of Medicine
University of New Mexico School of Medicine, Albuquerque, N.M.

11:15-12:00 Noon Questions and Answers
Drs. Seller, Gifford & Whang

12:00-2:00 p.m. Lunch break

2:00 p.m. House of Delegates (Final Session)

Cryptococcosis: A Case Report and Review

Capt. Richard Hess, MC*, *El Paso*

Cryptococcosis (Turolosis) is a mycotic infection caused by *Cryptococcus neoformans*; it may assume several clinical configurations. Pulmonary lesions usually precede other sites of involvement; lung involvement may heal without further progression and may even pass unrecognized. Dermal, skeletal and visceral lesions may occur during dissemination of the disease, but involvement of the central nervous system with subacute or chronic meningitis is the most familiar form of systemic torulosis. This is usually the cause of death.

The tendency of cryptococcosis to coexist with other diseases is well known. Torula infection occurs most often with Hodgkin's disease, lymphosarcoma, leukemia, tuberculosis, diabetes, sarcoidosis and steroid therapy. In cryptococcal meningitis concomitant (predisposing) disease is present in 50 per cent of the cases.[1,2,3]

It is the purpose of this communication to report our experience with a patient with cryptococcosis and to review some of the current knowledge concerning this apparently ubiquitous mycotic infection.

Case Report

V.B., a 22 year old Caucasian man was admitted to William Beaumont General Hospital because of hypertension. Four years earlier he was admitted to another hospital because of an abnormal chest radiograph. Sarcoidosis was confirmed after histologic examination of a scalene node. At that time he was asymptomatic but his chest radiograph showed progressive hilar adenopathy and parenchymal infiltration. Gradually he developed symptoms of respiratory insufficiency and lost weight. One month before admission he was noted to have hypertension which prompted his subsequent admission.

Physical examination revealed an asthenic Caucasian man in no distress. Blood pressure was 150/100; pulse rate, 76. Positive findings included a questionable blurring of the left optic disk and generalized lymphadenopathy. All else was negative.

Initial pertinent laboratory data included persistently elevated serum calcium levels, normal determinations of serum phosphorus and alkaline phosphatase. The blood urea nitrogen was between 25-40 mg/100 ml. Urinalysis showed numerous WBC's, RBC's and hyaline casts. Urine cultures were negative. An excretory urogram demonstrated diffuse nephrocalcinosis and nephrolithiasis; chest radiograph showed marked hilar adenopathy and diffuse parenchymal infiltration. A tentative diagnosis of sarcoidosis was made.

The early hospital course was uneventful. Oral prednisone, 40 mg a day, yielded normal urine and serum calcium determinations. He remained asymptomatic for 18 days after instituting corticosteroid therapy; suddenly he developed a severe generalized headache. Physical examination revealed bilateral papilledema. Careful lumbar puncture demonstrated a very high CSF pressure, glucose 6 mg/100 ml and protein 56 mg/100 ml. An India ink preparation demonstrated *C. neoformans;* subsequent cultures were confirmatory. The patient was treated with intravenous and intrathecal amphotericin B (Fungizone); response was dramatic. Fifty-two days after instituting amphotericin B therapy, the patient's CSF studies were completely normal. He was discharged.

Discussion

Cryptococcosis is caused by a single species, *Cryptococcus neoformans.* The organism is a spherical yeast-like, non-mycelial fungus measuring 4-20 micra in diameter and possessing a thin wall. It is surrounded by a wide, gelatinous polysaccha-

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ride capsule the size of which varies from strain to strain but which may equal in thickness the diameter of the cell. Reproduction is by budding, and the buds are attached to the parent cell by a thin wall and narrow pore. On Sabouraud's glucose medium soft, mucoid cream-colored colonies are formed within a few days when incubated at 37°C.[4]

Sanfelice first isolated the etiologic yeast from peach juice in 1894. At about the same time Busse and Buscke reported isolation of the same fungus from a sarcoma-like lesion of the tibia and from dermal and other lesions in man. Von Hauseman in 1905 was apparently the first to observe the fungus in meningitis, but it was Versé in 1914 who recognized the first antemortem case of torula meningitis.[4]

Cryptococcosis has no respect for age, sex, race or occupation. The disease is world-wide in distribution. In 1950 Emmons isolated *C. neoformans* from barnyard soil and in 1955 he reported the frequent saprophytic association of cryptococcosis with the excreta of pigeons. Virulent strains of *C. neoformans* were isolated from pigeon manure found in old nests or under roosting sites on the upper floors of buildings, window ledges and similar locations in cities and in stables or barns in rural areas. Pigeon manure and contaminated soil are the important reservoirs and infection is thought to occur from airborn dissemination of the organism. Animal to man or man to man contagion is unknown.[4]

All races are susceptible; men are infected twice as often as women (factor of occupational exposure). All ages are vulnerable; most cases occur in 20-40 age group.

C. neoformans has been isolated as a cause of epidemic bovine mastitis in cattle from contaminated milking machines and antibiotics. This organism also attacks cats, dogs, horses, monkeys and other animals.

Clinically, cryptococcosis may be classified by the organ system involved, e.g. pulmonary, central nervous system, dermal, osseous and other visceral types. As of 1963, the literature suggested that 90 per cent of cases of cryptococcosis involved the central nervous system. The remaining 10 per cent involved lungs or other soft tissue structures.[5] However, this concept is changing, and with the recognition of more cases of pulmonary torulosis, meningeal involvement may become the exception rather than the now accepted rule.[6]

Primary pulmonary cryptococcosis has been said to be rare. However, recent reports suggest that pulmonary involvement is not uncommon and indeed may be the only manifestation of the disease. It has been estimated that 5-15,000 cases of subclinical or clinical pulmonary cryptococcosis exists each year in New York City alone.[6] Clinically, the pulmonary disease may vary from essentially asymptomatic to acute pneumonitis. Roentgenographically, the pulmonary disease may be classified as follows: (a) Single or multiple mass lesions, which may be as large as 10 cm in diameter and which may cavitate; (b) Pneumonic infiltrates, single or multiple, patchy or confluent that closely resemble other fungus disease or tuberculosis; (c) Multiple nodular densities of miliary or slightly larger than miliary size frequently associated with linear infiltrates which may closely resemble miliary tuberculosis, sarcoid or other fungal disease; (d) Cavitary lesions[7], hilar masses, extensive pleural reaction with or without effusion and calcifications are being reported with increasing frequency.

The incidence of pulmonary cryptococcosis is not known, and the lack of a reliable skin test and serologic techniques further impedes epidemiologic studies. Diagnosis is usually difficult for the same reasons, and also because attempts to culture the organism from sputum or biopsy materials is difficult. Animal passage through mice is often needed to demonstrate viable pathogenic cryptococci. It is probably correct to suspect the possibility of pulmonary cryptococcosis in any patient in whom a persistent, asymptomatic parenchymal lesion with inflammatory characteristics proves resistant to diagnosis by accepted laboratory procedures.[6] The duration of the pulmonary disease may be difficult to assess in the asymptomatic patient and it is not generally appreciated that the course of pulmonary cryptococcosis may be protracted, that the disease may remain stable for long periods of time, and that it may progress or regress very slowly.[7]

Central nervous system cryptococcosis has been the subject of extensive reviews.[1,2,8] Before the introduction of amphotericin B in 1956, cryptococcal meningitis was almost always fatal; 75 per cent of patients died during the first year of illness. Occasionally, progression of the disease was interrupted by temporary remissions; nevertheless, sur-

vival for longer than three years was unusual.

Presenting symptoms are variable. However, headache is by far the most common complaint; this is most frequently localized in the frontal and temporal region, but occasionally it is occipital or generalized. The following is a list of the frequency of presenting complaints among 40 patients with cryptococcal meningitis: headache, 29; mental changes, 18; visual changes, 16; nausea and vomiting, 13; pain or stiffness of neck or back, 13; chills or fever, 12; lethargy, weakness and fatigue, nine; ataxia, eight; aphasia or slurred speech, five; paresthesias, four; other (seizures, paresis, weight loss, incontinence, tinnitus, dizziness and abdominal pain), 13; no symptoms in six.

Cerebrospinal fluid findings at the time of diagnosis revealed abnormalities in cell count in 97 per cent, in protein in 90 per cent, and pressure was elevated in 64 per cent. Sugar values were abnormally elevated in 55 per cent. The predominant cell type was the lymphocyte. Sugar levels were less than 40 mg per cent in only 55 per cent of cases and less than 10 mg per cent in 13 per cent. Initial cell counts were lower in patients with diabetes and other pre-existing diseases. Yeast-like forms were seen on direct examination of cerebrospinal fluid in 57 per cent. *C. neoformans* was cultured from the CSF in 95 per cent, from urine in 37 per cent, blood 25 per cent, stool 20 per cent, sputum 19 per cent and bone marrow in 13 per cent. (Bone marrow cultures in histoplasmosis have been reported to be positive in 95 per cent.) Coexisting disease occurred in 20 of the 40 patients, diabetes mellitus in eight, Hodgkin's Disease in five and sarcoidosis in two. Single cases of myeloid metaplasia, silicosis, carcinoma of the breast, idiopathic thrombocytopenic purpura, rheumatoid arthritis and rheumatic heart disease have occurred.[6]

Another form of central nervous system cryptococcosis long recognized but rarely reported is a circumscribed, nodular brain lesion without accompanying meningitis. Microscopically, the lesion is similar to that seen in the lung and the clinical picture simulates that of neoplasm.[9]

Dermal cryptococcosis is usually associated with a systemic infection and an antecedent respiratory infection. However, cases have occurred without a recognized pre-existing pulmonary lesion. The skin lesion may occur by contamination of abrasion with infected soil. Infection of the skin usually occurs on the face, beginning as an acneform, firm, nodular, painless eruption which may enlarge, become necrotic and ulcerate. The lesions resemble carcinoma, sarcoidosis, tuberculosis or other fungus infections.[4,8]

A total of 22 cases of osseous cryptococcosis are included in the world literature. Of these, only nine were localized to the skeleton and were not part of a generalized process. Roentgenographically bone lesions due to fungi often cannot be distinguished from those of tuberculosis and have been mistaken for sarcomata. Osteolytic lesions with very little associated sclerosis are formed; boney trabecula are destroyed and replaced by granulation tissue.[10]

Granulomatous lesions due to cryptococcosis have been reported in the liver causing hepatic failure, adrenal gland, producing adrenocortical insufficiency, and in the prostate, resulting in frequency, urgency, dysuria and urinary retention.[11]

One other cryptococcal infection of interest is the generalized disseminated form which has been found in the newborn. Symptoms date from birth and include CNS manifestations, jaundice, hepatomegaly, splenomegaly, chorioretinitis and intracranial calcifications. Hydrocephalus and cerebral degeneration may occur. The manifestations resemble those of congenital toxoplasmosis and cytomegalic inclusion disease.[12]

Cryptococcal endocarditis is a rare event reported following cardiac surgery. Lesions of the eye include papilledema, uveitis, retinitis and keratitis.[4]

Diagnosis

The diagnosis of cryptococcosis depends on the recovery and identification of the organism. There are no dependable skin tests or serologic methods.

In dried, heat-fixed and stained films of pathogenic specimens, cells of cryptococcus collapse, stain deeply or erratically and may be unrecognized. The large size of cryptococcus (4-20 μ) permits its detection in unstained preparations, particularly if the mucoid capsule is demonstrated by an appropriate mounting fluid such as India ink.

To study spinal fluid, a drop of India ink is placed on a clean slide and mixed with a drop of spinal fluid. Under reduced light, the sphericle

cells of *C. neoformans* and their enveloping capsules are sought. If the fungus is not found upon immediate examination, centrifuge the spinal fluid at 3,000 rpm for 10 minutes and carefully withdraw the sediment with a pipette for microscopic examination and culture.

Sputum or pus should be mixed with 10 per cent sodium hydroxide before examination. The capsule is resistant to sodium hydroxide, whereas pus cells and cellular debris will be destroyed.

Direct culture is performed by spreading material on neopeptone-glucose agar, Sabauraud's glucose agar and other media containing one to two per cent sugar and then incubating at 37°C. Cryptococcus is isolated with difficulty in some cases; therefore it is important to plant a large volume of fluid (2-5 ml).

Animal inoculation may be helpful; the organism has been recovered after passage through mice when it was not found on direct culture. Laboratory animals are susceptible to experimental cryptococcus, and a predictable form of the disease can be produced in mice.[4]

Effective therapy for disseminated and meningeal cryptococcosis has existed only since the introduction of amphotericin B in 1956. Optimum therapy requires the use of at least two gms of amphotericin B by the intravenous route.[1,2,3] Supplementary amphotericin B by the intrathecal route has not altered the morbidity or mortality of meningeal cryptococcosis.[2] Intrathecal administration should never be used alone since many cases of meningitis are associated with systemic disease.[2]

Relapse of meningitis following treatment with amphotericin B is perplexing and frequent. The incidence has been found to be 46 per cent during the first year. The likelihood of relapse has not been related to length of illness before treatment, persistence of organism on direct examination or abnormal CSF values at the end of treatment, nor has the incidence of relapse been increased in those with coexisting disease.[2]

Therapy of localized lesions, such as pulmonary nodules, prostatic granulomas or osseous lesions requires surgical excision as well as intravenous amphotericin B. Due to the potential nephrotoxicity of intravenous amphotericin B, certain investigators have recently suggested that clinically stable pulmonary lesions do not require therapy and should only be carefully followed by clinical methods.[6,13] At the Mayo Clinic, patients with focal, essentially asymptomatic, nonprogressive pulmonary lesions are not treated but are carefully followed.[13] However, this facet of therapy remains unsettled. When long-term clinical evaluation is impossible, amphotericin B should be given to any person with proven cryptococcosis, active or apparently inactive.

Conclusion

A patient with sarcoidosis in whom corticosteroid therapy was complicated by the development of cryptococcosis is reported. The pertinent literature is summarized. The ubiquitous nature of the disease and the capability to obtain a cure with amphotericin B dictate the need for increased awareness, accurate and prompt diagnosis.

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Bibliography

1. Speckard, A., et al: Improved Prognosis of Cryptococcal Meningitis with Amphotericin B Therapy. Ann. Intern. Med *58*:66, 1963.
2. Butler, W. T., et al: Diagnostic and Prognostic Value of Clinical and Laboratory Findings in Cryptococcal Meningitis. N.E.J.M. *270*:49, 1964.
3. Harris, T. R., Blumenfeld, H. B., Cruthirds, T. P., and McCall, C. B.: Coexisting Sarcoidosis and Cryptococcosis. Arch. Intern. Med. *115*:637, 1965.
4. Emons, C. W., Binford, G. H., and Utz, J. P.: *Medical Mycology*, Philadelphia, Lea & Febiger, 1963, Pp 147.
5. Knudson, R. J., Burch, H. B., Hatch, H. B.: Primary Pulmonary Cryptococcosis. J. Thorac. Cardiov. Surg *45*:730, 1963.
6. Houk, V. N. and Moser, K. M.: Pulmonary Cryptococcosis, Must All Receive Amphotericin B? Ann. Intern. Med. *63*:583, 1965.
7. Cohen, A. A., Davis, A., and Finegold, S. M.: Muronic Pulmonary Cryptococcosis. Amer. Rev. Resp. Dis. *91*:414, 1965.
8. Lettman, M. L. and Fimmerman, L. E.: *Cryptococcosis*. New York, Grune & Stratton, 1956.
9. Werner, W. A.: Pulmonary and Cerebral Cryptococcosis Without Meningitis. Amer. Rev. Resp. Dis. *92*:476, 1965.
10. Morris, E. and Wolinsky, E.: Localized Osseous Cryptococcosis. J. Bone Joint Surg. *47*:1027, 1965.
11. Tellotson, J. R. and Lerner, A. M.: Prostatism in an Eighteen-Year-Old Boy Due to Infection with Cryptococcus Neoformans. N.E.J.M. *273*:1150, 1965
12. Nelson, W. E.: Pediatrics, Philadelphia, W. B. Saunders, 1960, Pp. 564.
13. Geraci, J. E., et al: Focal Pulmonary Cryptococcosis: Evaluation of Necessity of Amphotericin B Therapy. Proc. Mayo Clinic *40*:552, 1965.

Symposium on Tropical Diseases

William Beaumont General Hospital, El Paso

(Editor's Note: This is the third and final part of a Symposium on Tropical Diseases presented at William Beaumont General Hospital. The first two parts have been published in previous issues of SOUTHWESTERN MEDICINE.)

EOSINOPHILIC MENINGITIS

Maj. Darrell S. Buchanan, MC*

There are a number of reports of sporadic cases of meningitis or meningoencephalitis associated with an eosinophilic pleocytosis of the CSF. The most common cause of such findings is cysticercosis infestation. Echinococcosis may produce similar findings, and there are occasional reports of eosinophilic pleocytosis accompanying non-parasitic infections.

During the two-year period from March 1958-60, several hundred cases of eosinophilic meningitis were observed on the island of Tahiti. Clinically, these cases usually exhibited an insidious onset of headache (usually bitemporal), stiff neck and generalized, random, spotty paresthesias. There was little or no temperature elevation with the illness and most patients remained active. The duration of illness ranged from several days to several months and relapses after complete recovery were not uncommon. About five per cent of cases had a Bell's palsy which was usually unilateral and occasionally the first symptom of the illness. The only significant laboratory finding was a marked cerebrospinal fluid pleocytosis (usually over 100 cells/mm^3). In 85 per cent of the cases, 25 per cent or more of the CSF cells were found to be eosinophils.

A beautiful epidemiologic study was performed on these cases and appeared to incriminate the eating of raw Skipjack tuna. It was assumed that the etiological relationship would be secondary to infection with parasites infesting the tuna. Examination of Shipjack tuna for parasites revealed a high incidence of cestode larvae and occasional nematode larvae. No proof of parasitic involvement of the nervous system was found in any of the human cases. Other epidemics of a similar clinical syndrome have been reported from New Caledonia (1951-53), Panape (1946-48), and Saipan.

A possible clue to the etiology of this syndrome is provided by two patients who died in a mental hospital in Oahu, Hawaii, December 1951 and January 1960. Both of these patients had long histories of chronic brain syndromes, and both had an eosinophilic meningoencephalitis. The brain of one was found to be infested with *Angiostrongylus cantonensis* (Chen) Dougherty, a metastrongylid lung-worm of rats. Because of the histologic similarity of the second case, it was assumed that *A. cantonensis* was the most likely cause of that patient's disease, also. This parasite has been reported as causing human disease in two other instances. The rat is the primary host for *A. cantonensis* and terrestial mollusc are the secondary host. Study has shown the molluscan host range to be quite broad.

The Tahitian and Panapian population, which was involved in the epidemic of eosinophilic meningitis, does not usually eat terrestial molluscs and also eat so little raw green vegetation that it is difficult to imagine them getting a significant ingestion of such molluscs by accident. The etiology of epidemic eosinophilic meningitis remains a mystery at this point.

References

1. Rosen, L., Laigret, J. and Bories, S.: Observations on an Outbreak of Eosinophilic Meningitis on Tahiti, French Polynesia. Amer. J. Hygiene, *74*:26-42, 1961.
2. Rosen, L., et al: Eosinophilic Meningoencephalitis Caused by a Metastrongylid Lung-Worm of Rats. JAMA *179*:620-624, 1962.
3. Weinstein, P.P., Rosen, L., Laquer, G.L. and Sawyer, T.K.: Angiostrongylus Cantonensis Infection in Rats and Rhesus Monkeys, and Observations on the Survival of the Parasite in Vitro. Amer. J. Trop. Med. *12*:358-377, 1963.

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TROPICAL DIARRHEAS

Maj. Robert C. Zurek, MC*

Although many micro-organisms can produce diarrheal disease, there are four organisms which, in proper settings, have distinguished themselves by causing significant epidemic diarrheas. These are: Vibrio comma, Shigella, Salmonella, and E. histolytica. Except for Cholera, they are worldwide in distribution. Primitive sanitation, poor personal hygiene, and state of nutrition, all common to tropical environments, foster outbreaks of diarrhea caused by these organisms. With the expanding United States military commitments in tropical areas these diseases are of obvious importance. In addition, modern transportation allows exposure to many American tourists to the primitive sanitation and unfamiliar infections of "far off lands." A jet aircraft can bring the tourist home well within the incubation period of many "exotic diseases." His symptoms may baffle the physician unaware of this situation.

Cholera

Cholera is an acute infectious diarrhea caused by a comma-shaped, gram negative, motile aerobic bacillus — Vibrio comma. Although once a cosmopolitan disease, it is presently endemic only in Asia (India, China, Burma, Pakistan, and Thailand). In South Viet Nam, there were 20,186 cases reported in 1964. The incidence is also rising in other countries of the Far East.

The disease is acquired by the ingestion of contaminated water and food stuffs. Man is the only known reservoir for cholera. The disease spectrum may vary from an asymptomatic carrier state to a fatal fulminant diarrhea. The cholera organism elaborates a potent endotoxin and mucolytic enzyme thought to be important in the pathogenesis of the disease. The old concept that the "rice" in the characteristic watery stool represents shed colon mucosa is no longer true. More recent studies have shown the small intestine to be involved in a non-specific mononuclear inflammatory process; no slough occurs. There is no septicemic phase in this disease.

The primary difficulty in cholera is severe fluid and electrolyte depletion. Recent investigations have shown the cholera stool to be isotonic; it contains approximately:

Na	137 mEq/1
K	16 mEq/1
Cl	107 mEq/1
H CO_2	45 mEq/1

Little glucose or protein is lost, but fluid loss may reach 15-20 liters per day in a severe infection. The feces are watery, mucoid, and odorless. Occasionally, they are bile stained and contain hemoglobin. Vomiting and abdominal cramps may be prominent, but fever is rare. Clinical signs and fatalities in this disease are related to dehydration and acidosis, previous state of poor nutrition, and general health. Deaths among occidentals (except POW's) are rare.

Mild cases run a 3-10 day course; little if any therapy is required. The treatment of severe cases presents an exercise in fluid and electrolyte balance — this is the most critical element.

Although many treatment schedules and monitoring systems have been recommended, (i.e. basing fluid replacement on such parameters as hematocrits, plasma specific gravity or osmolarity), these are felt by many to be unnecessary. Initial fluid replacement should be given rapidly to replenish losses. Adequate hydration can be judged by urine volume output (except in the most severe cases), quality of the pulse and pulse pressure, facial appearance, and if possible, body weight. Thereafter, IV fluids should match measured output in the stool. The single drop of plasma into graduated $CuSO_4$ to determine sp.gr. is a simplified technique. Insensible losses are covered by an ad lib oral fluid program. A suitable IV fluid can be obtained by mixing two volumes of isotonic saline to one volume isotonic sodium bicarbonate or Na lactate. Intravenous potassium replacement is rarely necessary and generally can be given orally along with carbohydrate containing substances such as fruit juices.

A very interesting method for following fecal fluid losses has been suggested by Dr. Geenough of East Pakistan. The patient is placed on a cot that has a hole located under the patient's buttocks. Placed on the cot, a rubber sheet with a sleeve through the hole channels the stool into the bucket. This permits collection of total output and avoids contamination of the area. The bucket should have a capacity of seven liters and preferably should be graduated to measure volume. A dip stick can be calibrated to measure the volume if necessary. (In the primitive areas where epidemic Cholera is a problem such tecniques as this and the plasma sp.gr. may be of great value.)

As already mentioned, adequate fluid and electrolyte replacement is all that is necessary to assure a satisfactory outcome in most cases. The addition

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of tetracyline 100 mgm IV q 8 hrs or 500 mgm q 6 hrs p. o. for at least 48 hours has been definitely proven to shorten the course of the disease and eradicate the carrier state. I feel tetracycline should be used in every case.

Vasopressor agents, cardiac stimulants, and cholinergic drugs and steroids are not indicated, and may be harmful.

Complications:

1. Acidosis is recognized by profound hyperventilation and should be corrected by increasing the amount of $NaHCO_3$ replacement.
2. Renal failure is rare; pre-renal cases are most common.
3. Hypokalemia, hypoglycemia and hypocalcemia with tetany is rare.

Bacillary Dysentery (Shigellosis)

Bacillary dysentery is an acute infectious disease characterized by pyogenic inflammatory lesions of the colon. Cardinal clinical symptoms are diarrhea, tenesmus, and abdominal cramps. In severe cases, mucous and pus are found in the stools; blood may or may not be present. Vomiting and fever usually occur. Proctoscopic examination may reveal a granular inflamed mucosa that bleeds easily; it may be covered by mucous or pus and may have shallow ulcerations not unlike ulcerative colitis. The WBC may be normal or elevated.

Dysentery is at home in all countries of the world, but occurs in epidemics mainly in the tropics where sanitation is primitive. The causative bacteria are acquired most commonly through ingestion of contaminated foods, rather than water. The human carrier is the main propagator of bacillary dysentery.

The organisms involved are numerous; many strains of Shigella have been identified. These include: Shigella flexneri (common in tropics), Shigella boydii, Shigella sonnei (prevails in area of good hygiene and is more resistant to treatment). There are many types within each species.

Although these organisms are known to produce potent endotoxins, the role of these endotoxins in clinical disease is not known. The pathogenesis of the diarrhea is also uncertain.

The natural course in disease of average severity is 7-10 days. In severe epidemics in India, the mortality rate has been as high as 30 per cent. Important factors influencing the outcome of the disease are the general nutritional state of the victim at the time of infection and lack of adequate supportive therapy.

Treatment

As in all diarrheal states — the cornerstone of therapy is adequate fluid and electrolyte replacement. Antibiotics have lowered morbidity and mortality considerably. Antibiotic therapy shortens the duration of the diarrhea and reduces problems inherent in long term replacement of fluid and electrolytes.

In most text books, treatment recommended is sulfonamide drugs. However, there is evidence of increasing microbial resistance to the sulfa drugs. Either Sulfisoxazole (Gantrisin) or sulfadiazine 2-5 gm may be given initially, then 2 gm every 6 hours for not less than 72 hours, and not more than 10 days. The non-absorbable sulfa drugs are also effective.

When sulfa resistance is encountered, Tetracycline 1 gm stat and 500 mgm q 6 hours is equally as effective and preferred by some as a primary drug. It has recently been reported that all Shigella in Viet Nam were resistant to sulfa drugs and approximatly 75 per cent were resistant to tetracycline. Complete sensitivity was found only to neomycin, kanamycin, colistimethate.

Salmonelloses

Acute diarrhea caused by the various salmonella organisms are clinically indistinguishable from shigella diarrhea; however, systemic complications secondary to a bacteremia are more common with the salmonella group. Vomiting and constitutional signs may be more prominent. The leukocyte count is usually normal. The natural course of the disease generally lasts only four to five days. A post-infectious carrier state is rare.

Salmonellae, unlike shigellae, infect many animal species. The infection is acquired by the ingestion of infected water or foodstuffs. Poultry products (including chickens, ducks, turkeys, and eggs) are probably the greatest single source of infection. The role of the asymptomatic carrier may be somewhat overrated. It appears that chronic debilitating diseases and partial gastrectomy renders one more susceptible to Salmonella gastroenteritis. The diarrhea is the result of actual bacterial invasion of the intestinal mucosa. The terminal ileum and cecum are most severely involved. Diagnosis can be made only by stool culture.

The value of antibiotic therapy in the treatment

of acute salmonella diarrhea is controversial. Chloramphenicol 3-4 gms daily in divided doses for at least two weeks is recommended by most authorities. Some advocate the use of bactericidal agents such as neomycin sulfate, paromomycin (Humatin) or colistimethate. Recently ampicillin has been found to be very effective against salmonella organisms.

As in all bacterial diarrheas, those due to salmonella are self-limited. With proper fluid and electrolyte management, fatalities should be rare. Agents such as bismuth and paregoric add greatly to the comfort of the patient.

Amebiasis

The last tropical diarrhea which I would like to discuss is Amebiasis. The Endomoeba histolytica has several forms which are called "races" or types. There is still much discussion about the importance of the large and small varieties; the typical large *E. histolytica* seems to be the cause of classic amebiasis. Some authorities believe there are also nonpathogenic large *E. histolytica* strains, especially in cold or moderate climates. The designation *E. dispar* has been reserved for such harmless strains. The small types have been called *E. hartmanni,* and are believed to be nonpathogenic. Man is the principal host of *E. histolytica.* Cyst contaminated water and food are the important vehicles by which amebiasis is transported from man to man. The trophozoites will not survive passage through the stomach. Carrier food handlers are especially dangerous. Apparently malnutrition and monotonous starchy diets predispose to amebiasis and increase the severity of the infection.

Following ingestion, the parasite encysts in the intestine and aided by bacterial enzymes penetrates the mucosa of the colon. Sites of predilection are cecum, ascending colon, and rectosigmoid areas. Flask shaped submucosal ulcers are produced; perforation is uncommon.

Amebiasis results in chronic colitis with intermittent episodes of diarrhea. Fever and vomiting are absent unless secondary bacterial infection occurs. The diarrhea is rarely if ever as severe as in the bacterial dysenteries, but is generally more protracted. The stools in amebiasis rarely contain white blood cells; this may be helpful in distinguishing amebic from bacterial diarrhea.

Unlike the bacillary dysenteries, amebiasis is not a self-limited disease; chemotherapy is very important. Many programs have been proposed, all are highly effective. However, no absolute cure is available at present. Follow-up stool examination is mandatory; re-treatment is frequently required.

The following drugs have been recommended for the asymptomatic cyst passer and *mild* diarrhea:

1. Carbarzone 0.25 gm b.i.d. for 10 days
or 2. Diodoquin 650 mgm t.i.d. for 21 days
or 3. Chloroquin 0.5 gm b.i.d. for 2 days
0.5 gm o.d. X 12 days

In cases with severe diarrhea, emetine HCL 65 mgm deep subcutaneously daily for 4-10 days is effective. In either instance, an antibiotic should be used in conjunction with the above agents. Such therapy is recommended to lower the bacterial flora which facilitates bowel wall invasion. Recommended antibiotics are:

1. Tetracycline 500 mgm q 6 hrs X 10-14 days
or 2. Fumagillin 50 mgm/day X 14 days
or 3. Humatin 500 mgm q.i.d. X 5 days

Some authorities recommend multiple drug programs such as chloroquin and diodoquin in conjunction with tetracycline in an effort to reduce the relapse rate.

SCHISTOSOMIASIS

Capt. Vernon H. Shull, MC*

Schistosomiasis has been and still is a major world public health problem, second only to malaria and tuberculosis. Approximately five per cent of the world's population (over 100 million individuals) are infected by the three principle organisms causing schistosomiasis. The impact which this disease had on our military personnel in Leyte and other areas in the Far Eastern Theater during World War II, the prevalence of the infection among the large influx of Puerto Ricans in this country, and now again among our troops in the Far East, makes this a timely subject for review.

The schistosome parasite was discovered in 1851 by Bilharz; thus the name bilharziasis. Human schistosomiasis results from infection by one or more of three species of blood flukes: *Schistosoma haematobium, S. mansoni,* and *S. japonicum.* Other species of schistosomes may occur in lower mammals and birds and occasionally these may infect man, giving rise to swimmer's itch. The

*Senior Resident in Medicine

genus Schistosoma belong to the class Trematoda, a part of the large phylum of flatworms, the Platyhelminthes. Other members of the Trematode class include the lung fluke, *Paragonimus Westermani,* and the liver fluke, *Clonorchis sinensis.*

In evaluating patients who may possibly have schistosomiasis, it is extremely helpful to determine whether they have been to any of the known areas of endemicity. In general, *S. mansoni* is the only species infective to man in the western hemisphere. *S. haematobium* co-exists with *S. mansoni* in many areas in Africa and the Middle East. *S. Japonicum,* on the other hand, resides only in the Far East, including Japan, China, Formosa, the Philippines, and Thailand.

The pathogenesis of this disease is more easily understood by first reviewing the life cycle of these parasites. The adult worms of Schistosoma have separate sexes, unlike other Trematodes of man. The females of *S. japonicum* and *S. mansoni* deposit their eggs in small venules of the human intestinal wall. Some of these eggs are gradually extruded into the lumen and are discharged in the feces. *S. haematobium* eggs are usually deposited in the venules of the urinary bladder and are found in the urine. The eggs hatch within a few hours after dilution of the feces with fresh water, liberating a ciliated free-swimming larva known as a miracidium. Upon reaching an appropriate snail which can serve as intermediate host, the miracidium undergoes development and an asexual multiplication which requires several weeks. The ultimate result is the production of numerous free-swimming, fork-tailed cercariae, which is the infective stage for man. The cercariae penetrate the skin or mucous membranes, which in some instances is accompanied by pruritus and punctuate erythema. They then migrate through the lymphatic and venous systems to the lungs, left side of the heart, and general circulation, finally reaching the intrahepatic portions of the portal vein where they mature and pair. They then proceed against the blood stream to small branches of the mesenteric veins where they become established. The diseases produced by these flukes are very similar and differ only because of variations in the anatomical regions involved. The disturbances produced by *S. haematobium* arise mainly in the genito-urinary system, but the lower colon and rectum may be involved. Both *S. mansoni* and *S. japonicum* cause disturbances principally in the small intestine, colon and rectum. All three parasites may give rise to hepatic lesions, but severe cirrhosis, splenomegaly and ascites are more commonly the late results of infection with *S. mansoni* and *S. japonicum.* The last in general is the most severe and appears to be the most resistant to available chemotherapy.

The clinical picture of Mansoni schistosomiasis, for example, is so protean that most cases remain unrecognized unless stool examinations are studied specifically for ova. The acute phase of this disease may be similar to that of bacillary or amebic dysentery but generally this phase is mistaken for a mild, non-specific enteritis. The acute symptomatology usually occurs within three to eight weeks after the cercariae enter the skin. The acute symptoms may include variable degrees of allergic manifestations with eosinophilia, cough, wheezing, fever, abdominal cramps, diarrhea, hepatomegaly and splenomegaly. The intensity of the infection and condition of the host affect the course of the disease. As a rule, healthy, well-nourished individuals rarely develop significant symptoms. It is thought that some are immune to the disease. Experiments with animals show that immunity can be acquired. Pathologic alterations in the various organs are due to an allergic or foreign body type reaction, with fibrosis, intense eosinophilic infiltration, resulting in multiple pseudotubercles, cirrhosis, cor pulmonale or portal hypertension. Rarely the nervous system and skeletal muscles become involved. In the former case, symptoms may resemble those produced by a brain tumor. Seizures, paralysis, and myopathy have also been reported.

Diagnosis depends on the identification of the ova in the stools or rectual mucosa. Liver function tests are commonly abnormal and gammaglobulin is increased. An intradermal skin test evokes a reaction within 15 minutes and is positive in 98 per cent of proven cases. Serologic tests include various flocculation and complement fixation procedures as well as precipitation tests. The complement fixation test is most sensitive, being positive in at least 90 per cent of cases. However, neither the skin nor serologic tests give an indication of the activity of an infestation. The eosinophil count frequently parallels the intensity of the allergic response to thousands of eggs which become lodged in the intestines, liver, and lungs. Signs and symptoms in many cases are partially due to malnutrition and other parasitic infections. In several clinical reviews, death occured with, and not from, schistosomiasis.

Not one of the various drugs available for treating this infection is fully adequate. Curability seemingly depends mainly on the development of immunity. Some authors feel that all cases of *S. japonica* should be treated in order to prevent late complications. The only group of drugs which have shown reasonably good effectiveness for treatment of Schistosomiasis of all types are the trivalent antimony compounds, which include antimony postassium tartrate, tartar emetic), stibophen (Faudin); and antimony lithium thiomalate (Anthiomaline). A newer trivalent antimony compound, antimony dimercaptosuccinate, has not been fully evaluated in this country. However, its increased myocardial toxicity suggests that it offers no advantage over the other antimony compounds. However, British authors are most impressed with dimercaptosuccinate, and feel that this is the treatment of choice for *S. haematobium.*

Both the therapeutic effectiveness and toxicity of these drugs are due to antimony which poisons the gonadal organs of the adult worms to stop further egg production. In higher dosages, the drugs kill the worms themselves, which is a necessary aim of treatment since gonadal organs will regenerate within one to three months.

Toxicity is minor in 95 per cent of cases treated. This consists of paroxysmal coughing during intravenous injection, nausea, vomiting, toothache, abdominal pain, conjunctivitis, myalgia, arthralgia, bradycardia and electrocardiographic changes. The electrocardiographic changes consist of prolongation of the QT interval and depression or inversion of T-waves. These changes occur in up to 100 per cent of cases treated. Occasionally, "antimony hepatitis" develops, but is readily reversible on cessation of therapy.

Most authorities prefer the use of stibophen (Fuadin) for the treatment of *S. mansoni* and *S. haematobium.* The daily dose is 2-5 ml IM daily until a total of 90-100 ml is given. The cure rate approaches 100 per cent. For *S. japonicum,* tartar emetic is the drug of choice, and is given intravenously on alternate days. The total dose which will cure over 90 per cent of patients is 2.5 gm or 500 ml of the 0.5 per cent solution.

Coming Meetings

The Committee on Injuries of the American Academy of Orthopaedic Surgeons in cooperation with the Southwestern Medical School will sponsor a second Symposium on Trauma November 2-5, 1966, in Dallas, Texas.

Speakers at the comprehensive post-graduate course will be: Dr. Carroll B. Larson, Iowa City, Iowa; Dr. Joseph H. Boyes, Los Angeles; Dr. E. Burke Evans, Galveston; Dr. Marcus J. Stewart, Memphis; Dr. Sam W. Banks, Chicago; Dr. Ian N. MacNab, Toronto, Canada; Dr. Hanes H. Brindley, Temple, Texas; Dr. Edwin F. Cave, Boston; Dr. Vernon M. Bryant, Dr. Russel B. Graham, Dr. Harold H. Mattson, Dr. W. Kemp Clark, Dr. Edward W. Richardson, Dr. Marvin P. Knight, Dr. Livius L. Lankford, Dr. Leon F. Ware, Dr. Adolph H. Giesecke, Dr. G. Tom Shires, Dr. Charles R. Baxter, Dr. Robert N. McClelland, Dr. William A. Cook, Dr. Malcolm Perry, Dr. William C. Garre, Dr. Robert G. Grossman, Dr. Robert V. Walker, Dr. Jack Reynolds, Dr. Ronald C. Jones, Dr. G. Truett James, Dr. Louis H. Paradies, and Dr. L. Ray Lawson, all of Dallas, and Dr. Frank Parrish, Houston.

The Symposium will be held in the Marriott Motor Hotel. Dr. Charles F. Gregory of Dallas is Director. Registration is $75, which includes luncheons and one evening's entertainment.

American College of Obstetricians and Gynecologists, Houston, October 24-26, 1966.

American Association of Public Health Physicians, San Francisco, October 31-November 4, 1966.

Interim Meeting of the New Mexico Medical Society, Alamogordo, New Mexico, Holiday Inn, November 11-12, 1966.

Western Surgical Association, Phoenix, Del Webb's Townehouse, November 16-19, 1966.

Eighth National Conference on the Medical Aspects of Sports, in conjunction with the 20th Annual Clinical Convention of the AMA, Las Vegas, Nevada, Caesar's Palace, November 27-30, 1966.

Southwest Allergy Forum, Galveston, Flagship Hotel, January 19-21, 1967.

American Cancer Society, Dallas, Sheraton-Dallas Hotel, May 3, 1967.

American Gynecological Society, Phoenix, Arizona Biltmore Hotel, May 4-6, 1967.

Texas Medical Association, Dallas, May 4-7, 1967.

85th Annual Meeting of the New Mexico Medical Society, Santa Fe, La Fonda Hotel, May 18-20, 1967.

48th Biennial Meeting of the Southwestern Medical Association, El Paso, Sheraton-El Paso Motor Inn, February 8-10, 1968.

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November, 1966

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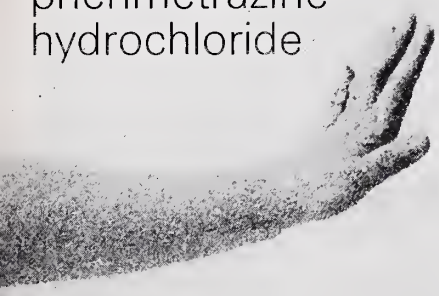

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*Barnes, R. H.: J.A.M.A. 166:898, 1958.

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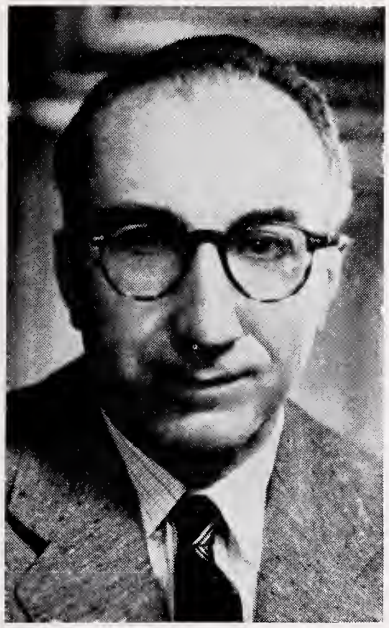

Dr. Michael E. De Bakey to Speak at Southwestern Medical Meet

Dr. Michael E. De Bakey, Professor of Surgery and Chairman of the Department of Surgery at the Baylor University College of Medicine in Houston, will be one of the speakers at the 48th meeting of the Southwestern Medical Association in El Paso, February 8-10, 1968, Dr. Laurance N. Nickey, El Paso, President-Elect and Program Chairman, has announced.

Dr. De Bakey is one of several outstanding speakers who will be brought here for the biennial meeting of the Association, which draws physicians from West Texas, New Mexico, Arizona, Nevada, and Northern Mexico. Translation facilities will be provided for the convenience of physicians from Mexico.

Dr. De Bakey in 1964 was Chairman of the President's Commission on Heart Desease, Cancer and Stroke. In 1965 he was a Director of Oak Ridge Associated Universities, a member of the Scientific Advisory Board of the Tulane University Delta Regional Primate Research Center, and a member of the Advisory Council of the University of Texas Graduate School of Biomedical Sciences. He has been Chairman of the Board of Regents of the National Library of Medicine. He is a member of the Program Planning Committee and Committee on Training of the National Institutes of Health.

He is a Past President of the American Association for Thoracic Surgery, the International Cardiovascular Society, the National Association on Standard Medical Vocabulary, the Society for Vascular Surgery and the Southwestern Surgical Congress. He has been a member of the Board of Governors of the Society for Cryobiology. He is a Director of the Association for the Advancement of Medical Instrumentation.

He was Editor of General Surgery Volume II, Medical History of World War II, and is Editor of the Year Book of General Surgery. He is a member of Editorial Boards of The American Surgeon, Annals of Surgery, Cardiovascular Research Center Bulletin, Circulation, Cryobiology, Heart Bulletin, Surgery, Postgraduate Medicine, Medical World News and the Journal of Cardiovascular Surgery.

Dr. De Bakey was born in Lake Charles, Louisiana, and received B.S., M.D. and M.S. Degrees from Tulane University. He interned in Charity Hospital in New Orleans and took Residencies in Charity Hospital, the University of Strasbourg and the University of Heidelberg. He was on the Tulane Faculty until 1948, when he took his present position.

Headquarters for the 1968 meeting will be at the Sheraton Motor Hotel in El Paso.

Dr. Frank A. Rowe of Albuquerque is President of the Southwestern Medical Association.

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Those Curious Globulins

MODERATOR: CAPT. NEIL A. KURTZMAN, MC, *Chief Resident in Medicine*

I. The Orthochemistry of Immune Globulins —
MAJ. WALTER J. DECKER, MSC

II. The Pathochemistry of the Immunoglobulinopathies —
MAJ. MARTIN L. NUSYNOWITZ, MC

III. Clinical and Pathogenic Immunoglobulinopathies —
CAPT. CHARLES P. CAVARETTA, MC

IV. Management of the Gammopathies —
CAPT. JEREMIAH J. TWOMEY, MC

The Orthochemistry of Immune Globulins

MAJ. WALTER J. DECKER, MSC

This presentation is concerned with the chemical composition and chemical reactivity of the two established classes of immune globulins, γ-globulins and macroglobulins.

The γ-globulins are arbitrarily defined as those serum (or plasma) proteins which move most slowly when submitted to electrophoresis at pH 8.6. It has been generally recognized that an association exists between γ-globulin and precipitating antibody; Grabar has suggested that all γ-globulin is antibody.[1] If this hypothesis is correct, then the physiochemical characterization of γ-globulin will also describe classic antibodies.

Deutch *et al* demonstrated that normal human γ-globulin could be divided into two distinct fractions upon the basis of significantly different mean electrophoretic mobilities.[2] They named these globulins γ_1 (I_gA) and γ_2 (I_gG). It was later demonstrated that these two fractions were themselves heterogeneous; immunoelectrophoresis studies have shown 18 lines of flocculation, indicating the presence of at least that number of subfractions.[3] Hence, it is evident that γ-globulin is composed of a large number of chemically distinct moieties. The difficulty in resolving, isolating, and chemically analyzing each of these compounds lies in the fact that their chemical and physical properties are very similar; they are all proteins of nearly the same molecular weight, amino acid composition, density, and electrophoretic mobility.

Reported mean molecular weights of normal human γ-globulins have ranged from 153,000-190,000.[4,5] These are the so-called 7S (I_gG) components; the variation in this range appears to be due to experimental error. No correlation has been made with species difference, nor do antibody type or content appear to have any bearing. Both ultracentrifugal and electrophoretic analyses of γ-globulin have demonstrated major, minor,

and trace components.

Another method of characterizing γ-globulin is ion-exchange chromatography. Using a diethylaminoethyl cellulose (DEAE) column, Sober and Peterson found two major and four minor peaks; the latter were associated with other globulins. Later, these investigators attained some separation of different antibodies with carboxymethylcellulose (CMC) chromatography.[6] Partition chromatography has been used with some success; antibodies for specific antigens appear in the eluate in a complex manner; at first they appear in the slower moving fractions, later some elute in the middle fraction, and finally they are almost entirely contained in the middle fraction. Porter proposed that this complexity could be due to any of several reasons: (a) different cells could produce slightly different γ-globulins, (b) a feedback γ-globulin synthetic system could be operable such that changes in environment could result in small changes in γ-globulin, and (c) the majority of the γ-globulin molecules could assume the most stable configuration under stress (Pauling's theory).[7] *In vitro* studies, using radioactive amino acids, have shown that different tissues label the fractions at different rates; hence, hypothesis (a) seems to be the most likely of the three.

Amino acid residue analyses have been quite disappointing; no significant differences have been noted between antibodies in a given species. Studies have shown that human γ-globulins from normal sera have one N-terminal aspartic acid residue and one or two N-terminal glutamic acid residues.[8,9] No correlation exists between the N-terminal amino acid and the γ-globulin's mobility. It is interesting to note that γ-globulins from pathologic sera vary considerably in both number and type of their N-terminal amino acids.[10] Serine and glycine were the two principal C-terminal amino acids found in normal human γ-globulins.[9]

Rosevear and Smith have shown a glycopeptide present in human γ-globulin; they determined its structure as Glu.Glu.Asp.(NH_2)-Tyr.Glu.Asp.(carbohydrate).[11] The carbohydrate moiety consisted of hexose, eight moles; glucosamine, six moles; fucose, two moles; and sialic acid, one mole.

It is believed that internal disulfide bonds in γ-globulins are required for immunological reactivity; if these bonds are broken by reduction to sulfhydryl groups, up to a seven-fold decrease in binding power of purified antibody and its homologous hapten can be demonstrated.[12]

Enzymatic degradation studies have shown that human γ-globulin can be broken down into half and quarter molecules (molecular weights approximately 80,000 and 40,000, respectively). The half molecules often retain antibody activity,[13] which indicates that not all of the γ-globulin molecule is required for immune activity. Current opinion is that the rest of the molecule may be necessary for the proper fit of the antigen. Such varied compounds as polypeptides, proteins, polysaccharides, and even lipids (the latter proposed by Felsenfeld et al in borrelia studies,[14]) may require a microcosmic "ball park" for proper attachment to the γ-globulin. The valency, or number of combining sites per molecule of antibody is generally agreed to be two. Stolinsky and Fudenberg[15] have recently reported on univalent fragments of human 7S globulins.

Recently, an analysis of the amino acid sequence of a Bence-Jones protein was reported.[16] These proteins are light chains of myeloma globulin and are related to light chains of normal human immuno-globulins. From the products of enzymatic digestion, they have proposed a structure for Type I Bence-Jones protein. They concluded that there are many structural differences (amino acid interchanges) in the N-terminal half of a molecule, but only one in the C-terminal half, when data on several individuals' Bence-Jones protein were compared.

Macroglobulins are present in normal human sera. They are high molecular weight compounds with sedimentation rates between 17-20S. The normal 19S (I_gM) component is made up of two different classes of protein; one migrates electrophoretically as a γ_1-globulin, and the other as an α_2-globulin.

Normal γ_1-macroglobulins and pathologic Waldenstrom macroglobulins are apparently closely related; the carbohydrate contents of the two are very similar, about 10 per cent by weight.[17] Waldenstrom macroglobulins seem to be more homogeneous than the normal γ_1-macroglobulins (I_gM) upon the bases of ultracentrifugal and electrophoretic studies.

One striking feature of the macroglobulins is the wide range of electrophoretic mobility of these

substances. One class of pathologic macroglobulins, the α_2-myeloma proteins, migrates electrophoretically at a very fast rate. Recent preliminary experiments indicate that a macroglobulin found in sera from human cholera patients migrates at the same rate as the α_2-myeloma protein.[18]

Summary

A brief review of the chemical composition and reactivity of immune globulins has been presented. These compounds have been characterized by electrophoretic and ultracentrifugal analyses, ion-exchange chromatography, amino acid residue determinations, chemical reactivity studies, and enzyme-induced degradation analyses. Although the molecular structures of immune globulins have not yet been completely determined, enough information has been gathered to provide working models explaining the biological behavior of these entities.

References

1. Grabar, P.: Ann. Rev. Biochem., 19:453, 1950.
2. Deutch, H.F., Alberty, R.A., Gosting, L.J., and Williams, J.W.: J. Immunol., 56:183, 1947.
3. Poulik, M.D.: Nature, 180:1477, 1957.
4. Pedersen, K.O.: in Ultracentrifugal Studies on Serum and Serum Fractions, p. 162, Almquist and Wiksell, Uppsala, 1945.
5. Scatchard, G., Gee, A., and Weeks, J.: J. Phys. Chem., 58: 783, 1954.
6. Sober, H.A., and Peterson, E.A.: Fed. Proc., 15:116, 1958.
7. Porter, R.R.: Biochem. J., 59:405, 1955.
8. McFadden, M.L. and Smith, E.L.: J. Amer. Chem. Soc., 75:2784, 1953.
9. Lay, W.P. and Polglase, W.J.: Canad. J. Biochem. Physiol., 35:39, 1957.
10. Putnam, F.W. and Miyake, A.: Arch. Biochem. Biophys., 65: 39, 1956.
11. Rosevear, J.W. and Smith, E.L.: J. Amer. Chem. Soc., 80:250, 1958.
12. Karush, F., in Serological and Biochemical Comparison of Proteins, W.H. Cole, ed., p. 40, Rutgers Univ. Press, New Brunswick, New Jersey, 1958.
13. Petermann, M.L.: J. Amer. Chem. Soc., 68:106, 1946.
14. Felsenfeld, O., Decker, W.J., Wohlhieter, J.A., and Rafyi, A.: J. Immunol., 94:805, 1965.
15. Stolinsky, D. and Fudenberg, H.: Nature, 200:856, 1963.
16. Titani, K., Whitley, E. Jr., Avogardo, L., and Putnam, F.W.: Science, 149:1090, 1965.
17. Muller-Eberhard, H.J. and Kunkel, H.G.: Clin. Chem. Acta, 4:252, 1959.
18. Felsenfeld, O., Wohlhieter, J.A., and Decker, W.J.: unpublished observations.

The Pathochemistry of the Immunoglobulinopathies

Maj. Martin L. Nusynowitz, MC

The purpose of this portion of the discussion is to provide the conceptual bridge between the chemistry of the immunoglobulins and the clinical manifestations of the various immunoglobulinopathies. One must have a basic understanding of the alterations of the normal chemistry which occur in these disorders in order to understand both the pathogenesis of the manifestations of the clinical entities and their treatment.

General

The immunoglobulins (Ig) are a group of structurally related proteins whose main, if not sole, function is to act as antibodies. Each class of Ig is comprised of a large and indeterminate number of proteins sharing certain physico-chemical, immunologic, and structural properties, and possessing individually specific properties responsible for their antibody and antigenic specificity.

Table I depicts some of the chief characteristics of the immunoglobulins. It is to be borne in mind that all components are believed to be present in normal sera, including Bence-Jones protein. The quantities present may be so minute, however, that they are undetectable in most circumstances.

The majority of acquired antibodies are found among the IgG fraction; several antibodies, among them those to diphtheria and tetanus toxins, typhoid O, and parathyroid B, are members of the IgA fraction; saline isohemagglutinins, Rh, rheumatoid factor, cold hemagglutinins, and heterophile antibodies are located in the IgM fraction; ragweed binding antibody and diphtheria toxoid antibody are located in all three. IgM antibodies are commonly directed against complex antigens; they are found mainly in the early stages of primary immunization, and are followed by IgG antibodies, which are the only types elicited by secondary immunization.

TABLE I

Characteristics of the Immunoglobulins

Name	IgG	IgA	IgM	IgD	Bence-Jones
Synonym	7S, γ_2, γG	γ_{1a}, β_{2a}	Macroglobulin β_{2M}, γ_{1M}, 19S		Microglob.
Molecular Weight	150,000	150,000	1,000,000	?	22,000-44,000
$S_{20,w}$	6.6	6.6-13	18(24,32,195)	7	2.2, 3.4
Mobility	Mid γ	Slow β	Fast γ	Slowβ	$\gamma + \beta$
Carbohydrate	2.6%	10.7	12.2	?	O
Antibody	+	+	+	?	O
% of -glob.	71%	22%	7%	<1%	No %

Chemistry

IgG, IgA, and IgM constitute the three main types of Ig, and all three are composed of two kinds of polypeptide chains, each being produced under separate genetic control. The light chain is common to all three types; its molecular weight is approximately 20,000 and it occurs in two forms: *K* (I, K, B) *light chains* occur in 60 per cent of the molecules in each class of Ig, and *L* (II, λ, A) *light chains* occur in 30 per cent. Ten per cent are unclassified. Because the light chain is common to all three types of Ig, it is responsible for the immunologic interrelationship between all three types of Ig.

The *heavy chain* is the other polypeptide chain occurring in the Ig. This chain confers specificity to each of the immunoglobulins; hence, there is a heavy chain corresponding to each Ig: Hγ, Hα, Hμ, Hδ. Each of these may have sub types. The molecular weight of Hγ is about 60,000. Each immunoglobulin contains two heavy and two light chains; hence, IgG may be of the type γ_2, K_2, or γ_2, L_2.

IgD has been recently described and is believed to contain two heavy chains (Hδ) and two light chains.

The heavy and light chains are linked by disulfide bonds and can be separated by appropriate chemical means. Proper treatment will fragment immunoglobulin molecules. It has been determined that the Fc fragment (fast) is responsible for skin fixation, placental permeability, complement fixation, fixation to microsomes, and Gm specificity, but possesses no antibody activity, while the Fab (slow) fragment possesses antibody activity.

The immunoglobulinopathies may be grouped into two groups of disorders: The hypogammaglobulinemias — comprising those conditions in which there is a diminution in synthesis of one or more of the immunoglobulins — and the hyperglobulinemias (paraproteinemias) in which there is an increase in production of one or more of the components, because of a neoplastic proliferative disorder of plasma cells and lymphocytes, which synthesize all immunoglobulins.

Hypergammaglobulinemias

Hypergammaglobulinemias, in turn, may be classified into two groups: (1) an essentially homogeneous protein product is synthesized presumably by a single clone of cells and (2) a variety of clones of cells proliferate, each producing an electrophoretically different γ-globulin.

The first type of hypergammaglobulinemia results from a proliferative disorder, usually considered malignant, in which one finds large amounts of homogeneous protein. These proteins may be closely related to one of the complete immunoglobulins or one of their structural subunits. Included in this group of disorders is multiple myeloma, macroglobulinemia, and heavy chain disease. The protein produced in large amounts is structurally similar to the normal corresponding immunoglobulin or Ig subunit, but exhibits greater homogeniety and lacks, for the most part, discernable antibody activity. Whether the protein itself is abnormal or rather represents normal protein produced in abnormal quantities is a still unsettled question.

In this category, the following disorders may occur.

1. Synthesis of light chain and one class of heavy chains is balanced. This results in complete IgG, IgA, IgD, or IgM. Thus, one sees as a result multiple myeloma with G, A, or D myeloma proteins, or macroglobulinemia.

2. Excess production of free light chains (either K or L types), resulting in multiple myeloma with only Bence-Jones proteinemia.

3. Combination of 1 and 2, resulting in multiple myeloma with abnormal serum immunoglobulin (as IgG or IgA) and Bence-Jones proteinemia.

4. Excess production of free heavy chains, resulting in Franklin's disease. Only one type has been described, viz.: type G in which one finds $H\gamma$. Type A, M, and D are postulated to occur.

It is of interest that the various types of immunoglobulins occur in multiple myeloma in about the same proportion as they do in normal sera. Thus, 54 per cent of patients with multiple myeloma in one series had IgG, and 22 per cent had IgA. IgD in multiple myeloma is rare. Twenty-three per cent of patients had Bence-Jones proteinuria alone. About 30 per cent of patients with G myeloma, and A myeloma, had Bence-Jones protein in addition. Similarly, 60 per cent of G myeloma is of the K, and 30 per cent of the L form.

Para-amyloidosis is a dominant feature when the only determinable abnormality is Bence-Jones proteinuria, Bence-Jones proteinemia, and decreased gamma globulin. It is thought that Bence-Jones protein may have a particular affinity to bind to certain tissue constituents (such as proteins or polysaccharides) to produce an insoluble proteinaceous infiltrate.

In all of these types of hypergammaglobulinemias, normal antibody production is defective.

The second category of hypergammaglobulinemias results from proliferation of a variety of clones of cells, and is characterized by excess IgG, IgA, or IgM in varying proportions. These may arise idiopathically or in association with varying diseases, such as systemic lupus erythematosis, ulcerative colitis, cirrhosis, etc.

Many of the clinical features of the various hypergammaglobulinemias are dependent on the particular immunoglobulin overproduced. The renal functional impairment produced by precipitation of Bence-Jones protein in the tubules, the coagulation defects in multiple myeloma and macroglobulinemia resulting from reaction of the proteins with specific coagulation factors, the circulatory impairment secondary to increased viscosity or cold insoluble globulins, (cryoglobulins) and the para-amyloidosis of Bence-Jones proteinuria will be discussed in a later section.

Hypogammaglobulinemias

We now turn our attention to the other end of the spectrum, and discuss those conditions in which there is a deficiency of immunoglobulin.

The standard classification of these disorders follows:

1. Transient — in infancy.
2. Symptomatic — associated with malignancy (as lymphoma or Chronic Lymphocytic leukemia).
3. Idiopathic
   a. Congenital (but not necessarily hereditary).
   b. Acquired (but not necessarily non-hereditary).

A simpler classification is that of Fudenberg and based on the chemical findings:

1. Typical — in which all 3 major immunoglobulins are severly depressed (75%).
2. Atypical — in which one or two immunoglobulins are depressed severely and the others are normal or increased.

An understanding of these conditions is dependent on an understanding of the genetic control of the production of the immunoglobulins. Human gamma globulin contains hereditary genetically determined antigens (Gm factors) located on its molecule. These factors are determined by several sets of allelic genes located on the chromosomes and inherited independently of one another. They are Gm_1, InV (Gm_2), Gm_3, and Gm_4. The alleles of the Gm_1 locus are Gm a (a family of factors) and Gm b (a single factor). Hence, in regard to Gm_1 factors, one may have three groups of genotypes, and their corresponding phenotypes, as determined by hemagglutination inhibition.

Gm^a / Gm^a	Gm (a + b −)
Gm^b / Gm^b	Gm (a − b +)
Gm^a / Gm^b	Gm (a + b +)

Similarly, the Gm_2 locus may have either of two co-dominant alleles namely, InV^a and InV^b.

Gm_1 factors are located on the $H\gamma$ chain in the Fc fragment, while InV factors are located on the light chain. Gm_3 and Gm_4 factors are located on the $H\alpha$ and $H\mu$ chains, respectively.

Thus, where the gene controlling the production of Gm_1 is deficient, one gets a decrease in the $H\gamma$ chain. Similarly, a decrease in Gm_3 and Gm_4 results in diminution of $H\alpha$ and $H\mu$, respectively. Lastly, since light chains are common to all the immunoglobulins, a deletion in InV results in a depression of light chain formation, yielding in abnormally low levels of all three immunoglobulins. Any combination of the above described gene failures is possible, and is explained by close linkages between the genes. Classical congenital agammaglobulinemia, the disorder in which there is a deficiency of all three immunoglobulins, is a sex-linked illness. A mutation in the InV locus, which is autosomal in location, would not explain the sex-linked character of this state. Thus, it has been postulated that although the structural gene for InV is on an autosome, the regulator gene is on an X-chromosome, and that a defect in this locus produces the abnormality.

Although most authorities feel that defective or deficient plasma cells and lymphocytes are present in these conditions so that gamma globulins are not produced, Fudenberg suggests that the deficiency of plasma cells formed from primitive lymphocytes is a *concomitant of* rather than a necessary condition for agammaglobulinemia, since this differentiation requires the stimulus of the process of antibody formation.

References

1. Osserman, E.F., and Takatsuki, K.: Plasma Cell Myeloma: Gamma Globulin Synthesis & Structure. Medicine 42:357, 1963.
2. Tomasi, T.B., Jr.: Human Gamma Globulin. Blood 25:382. 1965.
3. Fudenberg, H.: Lecture at Univ. Calif. Med. Ctr., 1965.
4. Rowe, D.S., and Fahey, J.L.: A New Class of Human Immunoglobulins, I. A Unique Myeloma Protein, J. Exper. Med. 121:171, 1965.
5. Rowe, D.S., and Fahey, J.L.: A New Class of Human Immunoglobulins, II. Normal Serum IgD. J. Exper. Med. 121:185, 1965.
6. The Immunoglobulins, (Edit), NEJM 272:376, 1965.
7. Solomon, A., and Fahey, J.L.: Bence-Jones Proteinemia, Am. J. Med. 37:206, 1964.
8. Osserman, E.F., and Takatsuki, K.: Clinical and Immunochemical Studies of Four Cases of Heavy Chain Disease. Am. J. Med. 37:351, 1964.
9. Comings, D.E.: Congenital Hypogammaglobulinemia, Arch. Int. Med. 115:79, 1965.

Clinical and Pathogenic Immunoglobulinopathies

Capt. Charles P. Cavaretta, MC

The Immune System

According to Peterson, Cooper and Good, the immune system has its anlagen in "gut associated lymphoid tissue." This, in turn, differentiates into two peripheral systems. The first, the thymus dependent tissue, is responsible primarily for cellular immunity, that is, delayed allergy and homotransplant rejection. The second is the immunoglobulin producing system.

The Primary Hypogammaglobulinemias

Three varieties of primary hypogammaglobulinemia have been described, two congenital forms having their clinical onset within the first year of life and an acquired form which is usually clinically apparent after the 15th year of life.

The most common *congenital* form of hypogammaglobulinemia, referred to as the Bruton type, is genetically a sex-linked recessive trait and therefore found primarily in male infants. Recurrent bacterial infections appear in the second six months of life. Characteristically, there is a deficiency in all immunoglobulins; however, cellular immunity is intact. Pathologically there appears to be a deficiency in the immunoglobulin producing system; the thymus dependent tissue is uninvolved.

The more rare congenital hypogammaglobulinemia, the Swiss type, appears to be an autosomal dominant, making its clinical appearance shortly after birth. These infants are susceptible to not only bacterial, but also to viral and monilial infections. The blood is deficient in lymphocytes, the serum deficient in all immunoglobulins and

cellular immunuity is impaired. This then appears clinically and pathologically to be a deficiency in both of the immune system components.

Acquired hypogammaglobulinemia also appears to be genetically determined. Although the clinical manifestation of recurrent bacterial infections, usually pulmonary, can occur at any age, serologically and pathologically it resembles the Bruton congenital form, that is, a deficiency in the immunoglobulin producing system.

The Secondary Hypogammaglobulinemias

The secondary hypogammaglobulinemias result from either a decreased production of, or an increased loss of immunoglobulins. The former mechanism is operative in malignancies of lymphoid tissues, leukemias, myelomas, and related disorders. Clones of malignant cells, producing abnormal globulins, suppress normal immunoglobulin production and/or mechanically crowd out normal cells. In the nephrotic syndrome and exudative enteropathies, immunoglobulins are lost to the urine and gut respectively, resulting in a deficiency state. As with other hypogammaglobulinemias, infection is more common and may be the first clinical manifestation of the underlying condition.

The Selective Hypogammaglobulinemias

Clinically, patients with single or multiple immunoglobulinopathies either are asymptomatic or subject to recurrent infections. A deficiency in IgA may be clinically silent, yet when associated with ataxia telangiecasia, frequent sino-pulmonary infections are encountered. Those patients with a singular deficiency as IgG or multiple deficiencies of IgA and IgG or IgM and IgG usually display a susceptibility to bacterial infection from early life. These rare selective deficiency states remain poorly understood and complete clinical and pathologic correlation remains to be explained.

The Dysgammaglobulinemias

The dysgammaglobulinemias are a heterogenous group of diseases which are characterized by a quantitative increase and a qualitative decrease in immunoglobulins. In this group are included Waldenstrom's macroglobulinemia, multiple myeloma, amyloidosis, and Franklin's disease.

In *Waldenstrom's macroglobulinemia*, there is a proliferation of cells of the reticuloendothelial system and an excessive production of IgM. These result in the clinical picture of hepatosplenomegaly, lymph node enlargement, anemia, macrocryoglobulinemia and the hyperviscosity syndrome.

The hyperviscosity syndrome is usually associated with macroglobulinemia and occasionally with lymphomas and multiple myeloma. The globulins, large in size and number, cause an increase in blood viscosity, resulting in sluggishness of blood, vessel occlusion and coagulation defects. Hemorrhagic diatheses, in particular epistaxis, dominate the clinical picture. Funduscopically, sausage-like segmentation of the retinal veins are found. Simple determination of the serum viscosity with the use of a viscosimeter can be diagnostic of the syndrome.

The diagnosis of Waldenstrom's macroglobulinemia can usually be ascertained on the basis of the clinical picture and the protein electrophoretic pattern. The macroglobulins on paper electrophoresis appear as a dense, narrow gammaglobulin band. When the clinical picture is not characteristic, the use of refined techniques, acrylic gel, electrophoresis or ultracentrifugation can be diagnostic.

Multiple myeloma is a malignant neoplastic disease characterized by proliferation of abnormal plasma cells. Bone pain, its most frequent presenting symptom, is caused by the proliferating plasma cells involving the marrow containing bones, resulting in discrete bone osteolysis or diffuse osteoporosis. Recurrent infections, especially pneumonias, are common because of the decrease in normal immunoglobulins. The abnormal immunoglobulin produced is either an IgA or G, commonly demonstrated on paper electrophoresis as a dense band migrating between the beta and gammaglobulin fractions, or occasionally with the beta or gamma fractions. The Bence-Jones protein is found in the urine in approximately 30 per cent of cases. Currently, this protein is considered to be a globulin composed of the "light chain" of the immunoglobulins. This protein appears to be the most common cause of the renal failure seen in multiple myeloma, impairing function by its precipitation in renal tubules. Other clinical findings, including cerebral and peripheral nerve involvement, bleeding, and organomegaly appear to be a result of localized tumor, amyloid, or the hyperviscosity syndrome.

Amyloidosis is a disease characterized by the de-

position in various tissues of a glycoprotein immunologically related to normal gammaglobulin. Osserman believes that this accumulation is actually precipitated globulin (light chain) and refers to amyloidosis as gammaloidosis. On the other hand, amyloid may represent a fibrous protein produced as a part of a chronic inflammatory response. Primary amyloid may be seen with multiple myeloma and consists of infiltration of the heart, tongue, kidney, liver, spleen and peripheral nerves. It may appear as a familial disease, and is occasionally found as the cause of cardiac decompensation in elderly individuals. Secondary amyloidosis complicates chronic inflammatory conditions such as rheumatoid arthritis, tuberculosis and osteomyelitis.

Recently, a group of patients have been described who have a lymphoma, bone marrow plasmacytosis, eosinophilia, uveal and palatal edema. Peaks are seen on their urine and serum electrophoretic patterns which have been found to represent free heavy chain proteins. This disease is known as *Franklin's* or *Heavy Chain* disease.

The *cryoglobulins* are an interesting heterogenous group of proteins which have as their common denominator the ability to precipitate on cold exposure. They may be subdivided into four classes according to whether they form a precipitate or a gel, and according to molecular size, 7S (IgG) or 19S (IgM).

Clinically, on exposure to cold the patients with cryoglobulins will develop purpura, epistaxis, visual or hearing disorders, or chills and fever. These result from precipitation of the globulins in the blood vessels, resulting in mechanical vessel occlusion an dantibody-antigen reaction (the antigen being the precipitated cryoglobulins). *Secondary* cryoglobulinemia is seen with Waldenstrom's macroglobulinemia, collagen diseases, and certain infectious states (SBE, lues). In the *idiopathic or primary* cryoglobulinemias, no etiologic cause can be determined. The diagnosis of cryoglobulinemia can easily be made by overnight refrigeration of the patient's serum. The formation of a precipitate or gel in the absence of cold agglutinens is diagnostic of cryoglobulins.

References

1. Peterson, R.O., Cooper, M.D., and Good, R.A.: The Pathogenesis of Immunologic Deficiency Diseases. AJM 38:579, 1965.
2. Gitlin, D.: The Hypogammaglobulinemias. Disease-A-Month. May 1962.
3. Fahey, J.L.: Antibodies and Immunoglobulins. JAMA 194:141, 1965.
4. Wysocki, K., and Mackiewicz, S.: Familial Anomalous B2aGlobulin. Arch Int. Med. 116:351, 1965.
5. Bayd, E.D., Hagedorn, A.B., McGuckin, W.F.: Macroglobulins, Its Recognition and Treatment, JAMA 193:100, 1965.
6. Rosen, F.S.: Macroglobulins, NEJM 267:491, 1962.
7. Rosen, F.S.: Macroglobulins, NEJM 267:546, 1962.
8. Saifer, A.: A Simple Chemical Test for Distinguishing Myeloma Globulins from Macroglobulins. Jour. of Lab. and Clin. Med. 63:1054, 1964.
9. Fahey, J.L., Barth, W.F., and Solomon, A.: Serum Hyperviscosity Syndrome. JAMA 192:120, 1965.
10. Smith, E., Kochwa, S., and Wasserman, L.: The Hyperviscosity Syndrome in Multiple Myeloma. AJM 39:35, 1965.
11. Logothetis, J., and Silverstein, P.: Neurologic Aspects of Waldenstrom's Macroglobulinemia. Arch Neur. 3:564, 1961.
12. Magnusson, S., and Maloney, W.C.: Multiple Myeloma. D M April 1960.
13. Donnelly, W.J., and Crahn, E.P.: Extraosseous Manifestations of Multiple Myeloma. Med. Clin. North Amer. 49:229, 1965.
14. Ritzmann, S.E., and Levin, W.C.: Cryopathies: A Review. Arch. Int. Med. 107:186, 1961.
15. Osserman, Elliott F.: Amyloidosis: Tissue Proteinosis: Gammaloidosis. Ann. of Int. Med., 55:1033, 1961.
16. Briggs, S.: Amyloidosis. Ann. of Int. Med., 55:943 ,1961.

Management of the Gammopathies

Capt. Jeremiah J. Twomey, MC

There are three components to the therapy of the dysglobulinemias: (1) management of the immediate effects of the dysproteinemia, (2) specific therapy against the cells producing the abnormal protein and, (3) parenteral replacement of normal immunoglobulins.

The hyperviscosity complications of the dysglobulinemias constitute a therapeutic emergency. The critical threshold is reached when the viscosity of the serum exceeds that of water fourfold.[1] This is a function of molecular size rather than molecular concentration. It is aggravated by the inability of these larger molecules to disperse throughout the extravascular bed.[2,3] The restriction of these abnormal globulins within the circulation makes them more readily available for plasmaphoresis. Plasmaphoresis is a relatively simple blood bank procedure.[4] Plasma is separated from a unit of the patient's blood, which is then reconstituted with physiological saline and returned to the patient. Multiple plasmaphoreses may be needed daily at first but eventually, main-

tenance can be achieved on a less frequent basis.[1] The result can be monitored by the viscosimeter devised by Fahey.[5]

The temperature at which cryopathies produce symptoms bears a relationship to the concentration of the abnormal protein in the plasma, which is usually in excess of 22 mg per cent before producing symptoms.[1] Plasmaphoresis is not effective because cryoproteins are usually 7S globulins and are dispersed outside the circulation. Maintaining a high environmental temperature is the only available treatment.

Therapy against the underlying disease should be instituted without delay. These diseases include the lymphomas, chronic lymphatic leukemia, multiple myeloma and idiopathic macroglobulinemia. Cryoproteinemia is also produced by carcinomatosis, the collagenoses, and chronic bacterial infection including subacute bacterial endocarditis. A large percentage of both gammopathies remain idiopathic. However, the proliferation of reticulum cells tends to group the idiopathic cases with the lymphoproliferative disorders. In these instances, the choice of alkylating agents is not of great importance because they all depend on the same chemical radical for their pharmacological effect.[7] The ability to continue maintenance therapy with chlorambucil and cyclophosphamide give these agents some advantage. Phenylalanine mustard and uracil mustard have yet to prove themselves superior to other alkylating agents in the treatment of multiple myeloma. This disease remains resistant to therapy. Effective chemotherapy often eliminates the need for continuing plasmaphoresis.[4,8]

There is no effective remedy for disseminated carcinomatosis. The collagenoses are best treated with pharmacological doses of steroids, and chronic infections, such as syphilis, require specific antibiotic therapy.

The third aspect of management involves assisting the resistance to infection. The cellular immune defense mechanisms are frequently impaired due to malignant transformation and immunologically incompetent proteins often replace their normal immunoglobulins. Less than 200 mg per cent of total γ-globulin is usually clinically significant.[9] Higher values due to the presence of immunologically inert abnormal globulins may mask an actual immunoglobulin deficiency state. There are usually 19S gamma M globulins[10,11] which probably reflect a breakdown in the normal sequence of antibody response. M immunoglobulins usually initiate the response to antigenic stimulus, but it is the 7S gamma G globulins that provide the major immunological defense against infection.[12] In fact, the elaboration of gamma G globulins seems to reciprocally inhibit macroglobulin synthesis.[13] In the absence of gamma G, gamma M globulins continue to be produced. The practical significance of these observations is that these deficiencies of gamma G globulin are masked by the excess of gamma M and are not detected by routine paper electrophoresis.

There are three outstanding considerations in gammaglobulin replacement therapy: (1) Only humoral immunity is being assisted; defective cellular immune mechanisms are not improved. (2) Maintenance requirements depend on the survival time of each dose given. It has been shown that the rate of gammaglobulin metabolism is inversely related to the circulating level of gammaglobulin.[15] This causes a somewhat greater requirement of gammaglobulin in deficiency states. It is unfortunate that the catabolic processes cannot distinguish normal from abnormal proteins. (3) There are seven immunological varieties of gammaglobulin controlled by at least two genetic loci.[16] Antibodies are demonstrable following homologous injections except in the complete absence of detectable gammaglobulins prior to administration. The immunological significance of these immunoglobulin antibodies has not been determined. However, only 10 mild reactions occurred among 10,000 weekly injections of gammaglobulin.[9] These can, in rare instances, reach anaphylactic proportions. Evidence to date continues to support the value of gammaglobulin replacement programs.

It has been estimated that the half-life of gamma G is about 24 days. It is recommended to give 1 ml of hyperimmune globulin intramuscularly per 10 pounds body weight at monthly intervals. It must be remembered that these are preformed antibodies and are not made to order for every antigenic stimulus that the host receives.

References

1. Fahey, J.L., Barth, W.F., and Solomon, A.: Serum Hyperviscosity Syndrome. JAMA 192-464, 1965.
2. Smith, E., Kochwa, S., and Wasserman, L.R.: Aggregation of IgGglobulin in Vivo I. The Hyperviscosity Syndrome in Multiple Myeloma. Amer. J. Med. 39:35, 1965.
3. Fahey, J. L.: Antibodies and Immunoglobulins II. Normal Development and Changes in Disease. JAMA 194-255, 1965.
4. Solomon, A., and Fahey, J.L.: Plasmaphoresis Therapy in Macroglobulinemia." Ann. Intern. Med., 58:789, 1963.
5. Fahey, J.L.: Serum Protein Disorders Causing Clinical Symptoms in Malignant Neoplastic Disease. J. Chronic. Dis. 16: 703. 1963.
6. Ritzmann, S.E., and Levine, W.C.: Cryopathies: A Review. Classification; Diagnostic and Therapeutic Considerations. Arch. Intern. Med. 107:754, 1961.
7. Dameshek, W., and Gunz, F.: Leukemia, The Alkylating Agents. Grune & Stratton Pub., 2nd Ed., pp. 462.
8. Bayrd, E.D., Hagedorn, A.B., and McGuckin, W.F.: Macroglobulinemia, It's Recognition and Treatment. JAMA 193:724, 1965.
9. Squires, J.F.: Hypogammaglobulinaemia, Proc. Roy. Soc. Med., 55:393, 1962.
10. Fahey, J.L.: Antibodies and Immunoglobulins I. Structure and Function. JAMA 194:71, 1965.
11. Finkelstein, Martin S., Uhr, Jonathan W.: Specific Inhibition of Antibody Formation By Passively Administered 19S and 7S Antibody. Science 146:67, 1964.
12. Hong, R., Schubert, W.K., Perrin, E.V., and West, C.D.: Antibody Deficiency Syndrome Associated with Beta-2 Macroglobulinemia. J. Pediat. 61:831, 1962.
13. Barth, W.F., Asofsky, R., Liddy T.J., Tanaka, Y., Rowe, D.S., and Fahey, J.L.: An Antibody Deficiency Syndrome. Selective Immunoglobulin Deficiency with Reduced Synthesis of Gamma and Alpha Immunoglobulin Polypeptide Chains. Amer. J. Med., 39:319, 1965.
14. Robbins, J.B., Miller, R.H., Arean, V.M., and Pearson, H.A.: Successful Treatment of Pneumocystis Carnii Pneumonitis in a Patient with Congenital Hypogammaglobulinemia. New England J. Med., 272:708, 1965.
15. Fahey, J.L., and Robinson, A.G.: Factors Controlling Serum Gamma Globulin Concentration. J. Exper. Med. 118:845, 1963.

Coming Meetings

Western Surgical Association, Phoenix, Del Webb's Townehouse, November 16-19, 1966.

Eighth National Conference on the Medical Aspects of Sports, in conjunction with the 20th Annual Clinical Convention of the AMA, Las Vegas, Nevada, Caesar's Palace, November 27-30, 1966.

11th Annual Clinical Conference, The University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, December 1-2, 1966.

First Annual Meeting of the Society for Cryo-Ophthalmology, Las Vegas, Nevada, The Dunes Hotel, January 8-10, 1967.

Southwest Allergy Forum, Galveston, Flagship Hotel, January 19-21, 1967.

American College of Surgeons, Arizona Chapter, Tucson, Arizona Inn, January 20-21, 1967.

New Mexico Medical Society's Conference of County Medical Society Officers, Albuquerque, Sheraton Western Skies, January 21, 1967.

19th Annual Midwinter Radiological Conference, Los Angeles Radiological Society, Los Angeles, International Hotel, January 28-29, 1967.

TMA District 1 Meeting, Pecos, February 4, 1967.

21st Annual Symposium on Fundamental Cancer Research, The University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, February 27-March 1, 1967.

American Academy of Pediatrics, San Francisco, Hilton Hotel, April 3-4, 1967.

American Society of Internal Medicine, San Francisco, St. Francis Hotel, April 7-9, 1967.

Southwestern Surgical Congress, Phoenix, Del Webb's Townehouse, April 10-13, 1967.

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American Academy of Neurology, San Francisco, Hilton Hotel, April 24-29, 1967.

Arizona Medical Association, Phoenix, Townehouse, April 26-29, 1967.

American Cancer Society, Dallas, Sheraton-Dallas Hotel, May 3, 1967.

American Gynecological Society, Phoenix, Arizona Biltmore Hotel, May 4-6, 1967.

Texas Medical Association, Dallas, May 4-7, 1967.

85th Annual Meeting of the New Mexico Medical Society, Santa Fe, La Fonda Hotel, May 18-20, 1967.

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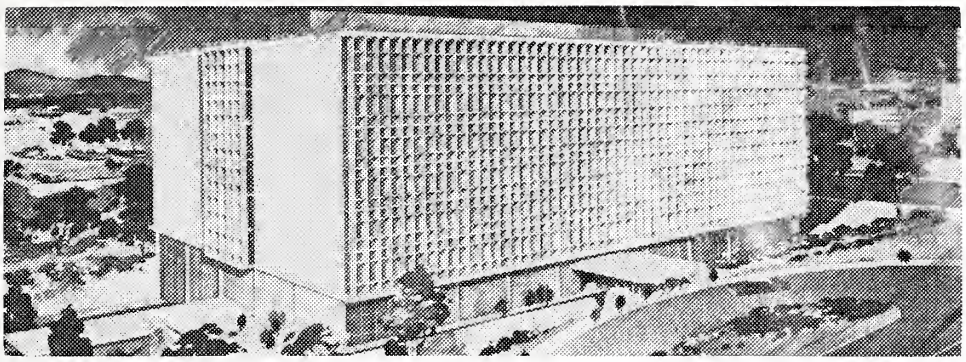

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What is a tranquilizer? According to the 24th Edition of Dorland's Medical Dictionary[3] a tranquilizer is "an agent which acts on the emotional state, quieting or calming the patient without affecting clarity of consciousness."

Defining a drug by its effects, however, can be misleading. The same effects by which the dictionary defines a tranquilizer have sometimes been seen after administration of a sedative — or, for that matter, a placebo.

Ambiguous though the term may be, it appears to be here to stay. The 1966 edition of the Physicians' Desk Reference[4] lists 42 tranquilizers indicated for treatment of anxiety and apprehensive states.

'Tranquilizers' have differences in action, differences in effect

Although all tranquilizers are intended to calm anxious patients there are differences in their actions — and in their effects. They have been divided into three categories — the rauwolfia group, the 'minor' tranquilizers, and the phenothiazines.[5]

Although the tranquilizing effect of rauwolfia has been known for centuries, its use as an antipsychotic agent in current practice has diminished.[5]

A 'minor' tranquilizer is often prescribed to achieve more than one effect. Thus, besides being tranquilizers some of these compounds may be muscle relaxants, antihistaminics with some calming action, anticholinergic sedatives, or antispasmodics.[5]

The phenothiazines are considered 'major' tranquilizers because they alter psychotic behavior.[1]

This classification may have done them more harm than good because it implies that the phenothiazines should be reserved for the more severely disturbed. This is not necessarily true.

The phenothiazines — and the problem of sedation

One of the problems of prescribing phenothiazines for ambulatory patients has been the fear that excessive sedation will impair the patient's ability to function. This, however, is less of a problem with some of the phenothiazines.

"Clinically they may be differentiated primarily in terms of their potency and the extent of their sedative effect, which appear to be inversely proportional. That is, the least potent, the one which is used in highest dosage, chlorpromazine, is the most sedative, while the reverse holds true for fluphenazine."[6]

In a recent report on various studies conducted over several years evaluating 360 patients treated for anxiety and stress with seven phenothiazines, this inverse relationship of potency to sedation was confirmed.[7] Also under consideration was the degree to which the particular phenothiazines neutralized anxiety (the angolytic index). Interestingly enough there was, again, an inverse relationship. The most sedative of the phenothiazines appeared to be the least active in neutralizing anxiety. Fluphenazine, one of the least sedative, on the other hand, was found to be most effective in relieving anxiety.[7]

RELATIVE SEDATIVE AND ANGOLYTIC INDICES OF PRINCIPAL PHENOTHIAZINES*

DRUG	SEDATIVE INDEX	ANGOLYTIC INDEX	BASED ON STANDARD DOSE OF
Chlorpromazine	100	15	25 mgs.
Triflupromazine	100	15	25 mgs.
Thioridazine	90	17	25 mgs.
Perphenazine	15	25	4 mgs.
Carphenazine	25	25	25 mgs.
Trifluoperazine	3.3	95	2.0 mgs.
Fluphenazine	3.5	100	2.5 mgs.

*adapted from Sainz[7]

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Precautions: Effects of atropine, anesthetics and C.N.S. depressants may be potentiated. Hypotension may occur in patients undergoing surgery. Do not use epinephrine for treatment of the hypotensive reactions which may appear in patients on phenothiazine therapy.

Side Effects: Extrapyramidal reactions, allergic skin reactions, the possibility of anaphylaxis, drowsiness, visual blurring, dizziness, insomnia, nausea, anorexia, salivation, edema, perspiration, dry mouth, abnormal lactation, polyuria, hypotension, and jaundice and biliary stasis may occur. Routine blood counts are recommended to determine possible blood dyscrasias; if symptoms of upper respiratory infection occur, discontinue drug and institute appropriate therapy.

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For full prescribing information, see package insert.

References: 1. Simpson, G.M.: Postgrad. Med. 39:557, 1966. 2. Freyhan, F.A.: Am. J. Psychiat. 115:577, 1959. 3. Dorland's Illustrated Medical Dictionary, ed. 24, Philadelphia, W. B. Saunders Co., 1965, p. 1603. 4. Physicians' Desk Reference, 1966, Oradell, N.J., 1965, p. 310. 5. Cohen, S.: Northwest Med.: 65:197, 1966. 6. Detre, T., and Jarecki, H.: Connecticut Med. 25:553, 1961. 7. Sainz, A.: Psychosomatics 5:167, 1964.

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IN THIS ISSUE

VOL. 47, NO. 12

December, 1966

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*Neyroud, M.: Praxis 44:648-650 (July 14) 1955.

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*Schiller, I. W., and Lowell, F. C.: New England J. Med. 261:478, 1959.

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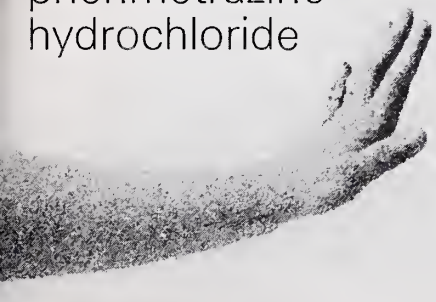

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Serum Lipids in Spanish- and Anglo-American Men*

Lora Mangum Shields, Ph.D., Carl H. Gellenthien, M.D.,
and Orhan M. Sansoy, M.D., *Las Vegas, New Mexico*

The mortality rate from coronary artery and hypertensive heart disease in New Mexico is approximately half that for the nation.[1] Records of 59,249 deaths ± during a 10-year period (1954-1963) relate this deviation in part to the New Mexico ethnic profile.[2] The New Mexico population consists predominantly of Spanish-and Anglo-Americans, with minorities of Pueblo Indians, Navajos and Mexican nationals. Death rates among these five ethnic groups differ significantly in (a) infant mortality, (b) deaths from cardiovascular diseases and (c) mortality of men and women for decades past 50.[2] Cardiovascular deaths, as a per cent of the ethnic population alive at the beginning of each decade, are approximately one-third higher in Anglo-American than in Spanish males during the fifties, sixties and seventies (Fig. 1). New Mexico, therefore, offers an appropriate laboratory for research into the influence of ethnic character on the incidence of cardiovascular deaths.

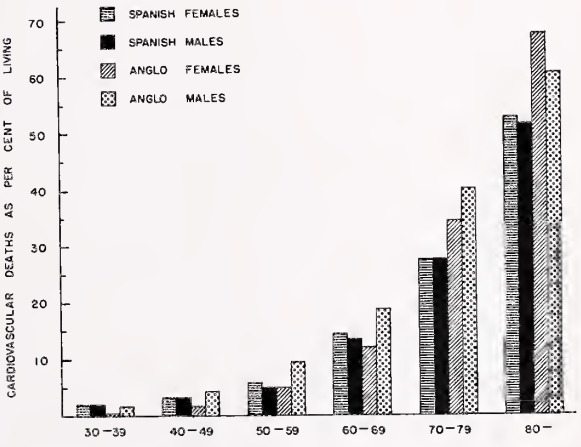

Fig. 1

For Spanish- and Anglo-American men and women, the percentage of deaths from cardiovascular disease compared to the living population by decade, based upon the number of individuals alive in each group at the beginning of the 10-year period.

An epidemiologic study was directed toward the relation of serum lipids implicated in cardiovascular disease and differences in cardiovascular deaths among males in New Mexico's two major ethnic groups. For 1094 Spanish-and Anglo-American men, serum fatty acids, triglycerides, cholesterol and phospholipids were measured in overnight fasting serum and serum drawn three hours after a high-fat test meal. Lipid concentrations were determined by Albink's procedures.[3] Cholesterol values for approximately 500 persons, however, were obtained on a Technicon autoanalyzer.[4] The fatty acids represent a summation of all serum lipids. Serum triglycerides and cholesterol have been causally related to atherogenesis. Triglycerides show a greater sensitivity than cholesterol to changes in fat metabolism, consequently may be of more value in diagnosing or in predicting a precoronary condition.[5]

From the New Mexico Highlands University, Valmora Sanatorium and New Mexico State Hospital.
* This study was supported by National Institutes of Health, grants He-06942, -02 and -03.
± These tabulations represent 34,242 Anglo-Americans, 19,117 Spanish-Americans, 1,435 Mexican nationals, 1,780 Pueblo Indians and 2,675 Navajos.

Phospholipids may be involved in the deposition of material within the blood vessel or in the clotting mechanism which initiates thrombosis.

Data for each ethnic segment were grouped by decade from 20 through 79 years. More than one-third of the test population were hospitalized mental or geriatric patients. The 330 apparently healthy men in the twenties were largely first-year university students boarding on campus. The two ethnic segments were well represented in both the normal and the institutionalized populations (Table 1).

Table 1. Ethnic Origin of Experimental Subjects

Ethnic group	Number	Non-hosp.	Hos.
Spanish-American	459	257	202
Anglo-American	635	468	167
Total	1094	725	369

Each individual of the non-student, apparently healthy population was presumed to be on a diet typical for his ethnic group. The diet for hospitalized patients averaged approximately 2000 calories daily. The test breakfast consisted of two eggs scrambled with 29 gm. of butter and 60 ml. of thick whipping cream. Each individual's fasting lipid concentration served as a control to the concentration after the test meal.

Results

A. Overnight fasting serum lipids for Spanish- and Anglo-American men in typical ethnic environments. — In a Spanish-American male population in a typical ethnic environment, serum fatty acids, triglycerides and cholesterol achieve maximum concentrations early in life, 10-30 years before the corresponding peaks in Anglo-Americans. Fatty acid concentrations are significantly greater in the Spanish during the thirties and in Anglos during the sixties (Fig. 2). Fasting triglycerides peak during the 30-through 39-year decade in the Spanish and during the forties in Anglos, preceding by about 20 years the peak incidence of atherosclerosis in the fifties and sixties and suggesting a causal relationship in the case of a chronic disorder which develops over a long period. [5] Cholesterol concentrations reach a maximum during the forties in Spanish-and during the sixties in Anglo-Americans. Phospholipids are highest in the sixties for apparently healthy individuals of both ethnic groups. Serum cholesterol and phospholipid concentrations are greater in the Spanish than in Anglos to age 50. Except for decades in which peaks occur for one ethnic group or the other, however, fasting fatty acids (Fig. 2), triglycerides cholesterol and phospholipids are not significantly different for the two.

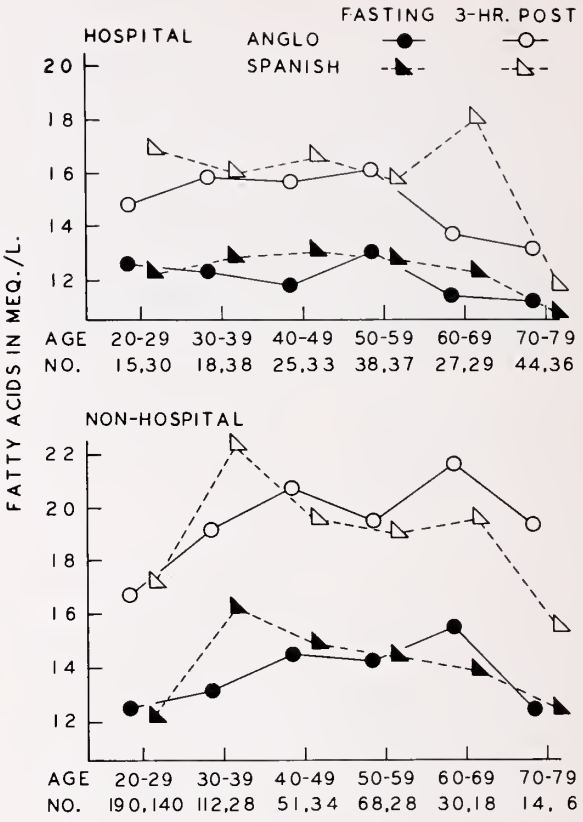

Fig. 2.

Comparison of means for fatty acids in fasting serum (solid symbols) and in the three-hour postprandial sample following a high-fat test meal (open symbols). Data are shown for Spanish- and Anglo- American men living at home in typical ethnic environments in contrast to institutionalized Spanish-and Anglo-American geriatric and mental patients. The number of individuals in each age group is shown to the right of No. below each graph, the first figure for each decade representing Anglos and the second, the Spanish.

B. Overnight fasting serum lipids for institutionalized Spanish and Anglo-American men. — In hospitalized mental and geriatric patients, overnight fasting serum lipid concentrations, with few exceptions, are lower than in the non-institutionalized. Peak concentrations for the two ethnic groups occur at more nearly the same age, later in the Spanish and earlier in Anglos than normal.

Serum triglycerides achieve a maximum during the fifties in institutionalized populations of both ethnic groups. Fasting fatty acids, cholesterol and phospholipids are highest in the hospitalized Spanish during the forties, compared with peaks during the fifties in Anglos. Only phospholipids are higher in hospitalized than in non-institutionalized men.

C. Three-hour postprandial serum lipids. — In serum drawn three hours after the test meal, peaks for lipids, other than phospholipids, tend to occur within the same decades as for fasting sera. Postprandial curves roughly parallel the fasting values. Elevations in lipids are greater in the non-institutionalized except during the twenties and during the sixties in the case of only fatty acids in Spanish men. (Fig. 2). This higher increment in fatty acids may reflect a coronary condition in a number of the Spanish men. Bronte [6] reported that after a meal containing a standard amount of fat, patients with ischemic heart disease show more intense and prolonged lipemia than normal. For the apparently healthy Spanish and Anglo men, fatty acid concentrations after the test meal are 20-40 per cent higher than in fasting serum, and triglyceride concentrations are 60-100 per cent higher. For the individual, cholesterol and phospholipids do not necessarily increase following a high-fat meal, but the average cholesterol concentrations increase by 12-30 mg. per cent.

The role of the stomach in fat digestion may be important to the time relationship in presenting material to the intestine. The inverse relationship between the degree of gastric acidity and the degree of lipemia after a fat-containing meal shows that individual variation in the ability to digest fat in a dose-response manner may determine blood lipid levels.[6] Oral administration of fat in other studies disclosed tolerance differences among individuals with and without ischemic heart disease, but intravenous administration failed to demonstrate any such differences.

D. Cardiovascular deaths and serum lipids by decade. — Peak lipid concentrations for Anglo males are somewhat lower than for Spanish men. The highest levels for Anglos, however, typically occur during the fifties and sixties, a period in which the cardiovascular death rate is increasing in this ethnic group. The peak in serum triglycerides is an exception, appearing 10-20 years earlier than maximum concentrations for other serum lipids. The greater cholesterol concentrations in the Spanish through the fifties are not associated with a higher incidence of cardiovascular deaths. Fatty acid (Fig. 2) and triglyceride concentrations, by decade, for apparently healthy Spanish-and Anglo-Americans, however, reflect the ratio of cardiovascular deaths to the living population. Cardiovascular disease is higher in the Spanish before and in Anglos after age 40, but for both ethnic groups constitutes an insignificant fraction of total deaths below 40 years (Fig.1).

The lower overall cardiovascular mortality for New Mexico may relate to a lower incidence of cardiovascular deaths in Anglo as well as among non-Anglo ethnic segments. Another relevant area which should be investigated is the effect of high altitude in promoting improved collateral circulation in myocardium.

Summary

In a male Spanish-American population in a typical ethnic environment, serum lipids achieve a maximum concentration early in life, 10-30 years before the corresponding peaks in Anglo-American men. Peak values are not significantly different in apparently healthy Spanish- and Anglo-American males, but concentrations in Anglos are greatest during the fifties and sixties when their incidence of cardiovascular disease is highest. In institutionalized mental and geriatric patients, maximum serum lipid concentrations in Spanish- and Anglo-American men occur in the same or adjacent decades, not showing the normal contrasts with age or ethnic division. The low serum lipid concentrations in Spanish men during the later decades of life, as well as the relatively low incidence of cardiovascular deaths in Spanish males, appear to identify the limited cardiovascular death rate in New Mexico in part with the ethnic profile.

References

1. U. S. Bureau of the Census, Statistical Abstracts of the United States, Eighty-fifth Edition. Washington, D.C. 943 p. U. S. Department of Commerce. 1964.
2. Shields, Lora Mangum and Naylor, Anita: Cardiovascular and Total Mortality Statistics (1954-1963) for Five New Mexico Ethnic Groups, Rocky Mountain Med. Jour. 63:54-57. 1966.
3. Albrink M. J.: The Microtitration of Total Fatty Acids in Small Amounts of Serum, with Notes on the Estimation of Triglycerides, Jour. Lipid Res. 1:53. 1959.
4. Leffler, H. H.: Estimation of Cholesterol, Amer. Jour. Clin. Pathol. 31:310-313. 1959.
5. Albrink, Margaret J., Meigs, J. W. and Man, E. B.: Serum Lipids, Hypertension and Coronary Artery Disease. Amer. Jour. Med. 31:4-23. 1961.
6. Bronte, B. Stewart: Fat Tolerance Studies in Ischemic Heart Disease. Geriatrics 19:554-561. 1964.

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The Nephrotic Syndrome

Moderator: Capt. Neil A. Kurtzman, MC

I. Etiology of the Nephrotic Syndrome —
Capt. Jeremiah J. Twomey, MC

II. Selected Clinical Aspect of the Nephrotic Syndrome —
Capt. Timothy Harris, MC

III. Pathogenesis and Laboratory Diagnosis of the Nephrotic Syndrome —
Capt. Neil A. Kurtzman, MC

IV. Pathology of the Nephrotic Syndrome (NS) —
Col. Robert H. Moser, MC

V. Treatment of the Nephrotic Syndrome —
Col. Robert H. Moser, MC

I. Etiology of the Nephrotic Syndrome

Capt. Jeremiah J. Twomey*

The nephrotic syndrome results from glomerular injury causing an increased permeability to protein and manifesting itself as proteinuria in excess of 3.5 grams daily per 1.73 M^2 body surface area. The sequellae of the protein loss include hypoalbuminemia, edema, hyperlipemia and fat bodies in the urine. It becomes apparent from the list of etiologies (Table I) that the nephrotic syndrome is an effect of injury to the nephron and is not itself a distinct disease entity. These are unlikely to be chance associations because only situations where the nephrotic syndrome bears a distinct temporal relationship to the associated disease or drug are included. A diversified list such as this demands careful scrutiny for common factors that may suggest pathogenetic mechanisms.

The majority of nephrotic syndromes are still "idiopathic". This is a reflection of current limitations of understanding of membranous glomerulonephritis. The frequency of lipid nephrosis in children and their responsiveness to steroids is analagous to leukemia and remains unexplained. Familial nephrotic syndromes[1] have occurred but the role of genetics has not been clearly established. Late recurrences after remissions lasting up to 15 years have been reported.[2]

The frequent association of the collagen diseases has implicated autoimmune disorders in the pathogenesis of the nephrotic syndrome. About half of

*Chief, Hematology Service

the patients with lupus have renal involvement but less than half of these develop the nephrotic syndrome.[3,4] In fact, it is an infrequent major manifestation of the disease.[5] The prognosis, when the nephrotic syndrome occurs, is not uniformly hopeless. Nevertheless, one-third of patients with this disease die from uremia. Serum sickness has also produced the nephrotic syndrome.[6] Nephrosis has been precipitated by hypersensitivity states such as bee stings.[7] Only half of four patients recovered despite the obvious limited exposure to the antigen. The impaired tubular reabsorption of filtered proteins contributes to the eventual proteinuria as well as the glomerular lesion. Poison oak produces both glomerular and tubular injury.[9]

Table I

Etiology of Nephrotic Syndrome

Idiopathic:	
Familial: (1)	
Collagen:	SLE (3-5)
	Henoch Schonlein (6)
	Scleroderma (6)
	P.A.N. (6)
Sensitivity:	Serum Sickness (6)
	Bee Sting (7)
	Pollen (8)
	Poison Oak (9)
Metabolic:	Diabetes Mellitus
	Amyloidosis (10)
Infections:	2° Syphilis (11)
	Leprosy (12)
	Tuberculosis (13)
	Malaria (14)
	S.B.E. (15)
Drugs:	Trimethadione 16)
	Phenindione (17)
	Probenecid (18)
	Penecillamine (19)
	Tolbutamide (20)
	Mercury (21)
	Gold (6)
Venous:	Renal Vein Thrombosis (22,23)
	Constrictive Pericarditis (24)
	Tricuspid Insufficiency (6)
Arterial:	Renal Arterial Stenosis and Nephrosclerosis (25)
Misc:	Sickle Cell Disease (26)
	Pheochromocytoma (27, 28)

The noxious agent seems to be a catechol-like compound. These hypersensitivity related cases are frequently associated with previous exposure to the same antigen and with sensitivity among immediate relatives. Amyloidosis has yet to be placed among the immune or the neoplastic diseases. Amyloid deposits in the glomeruli produce a relentlessly progressive type of nephrotic syndrome.[10] Perhaps diabetes and amyloidosis are best classified as a separate metabolic group. It is hard to segregate infection from altered immune mechanisms. The list of infectious associations with the nephrotic syndrome includes acute and chronic bacterial[11,13] and plasmodial[14] infections (Table I). Renal amyloidosis may confuse the etiology in leprosy.[12] Embolic injury must contribute in subacute bacterial endocarditis.[15]

The incidence of iatrogenic nephrosis is low but the list of incriminated drugs is likely to increase. The incriminated drug had been taken, in most instances, for a few months before the nephrosis developed. Only trimethadione nephrosis has been reproduced in experimental animals.[16] There have been isolated reports relating probenecid,[17] phenendione,[18] tolbutamide[19] and penicillamine[20] with nephrosis. Heavy metals, including gold and mercury, can produce the nephrotic syndrome which is often associated with a skin reaction. It is felt that this is a definite hazard of protracted mercurial diuretic therapy.[22]

The vascular etiologies remain apart from possible immune mechanisms. Best known are those conditions producing markedly elevated venous pressure in the renal parenchyma. Renal vein thrombosis may: (a) extend from the pelvic veins and the inferior vena cava, (b) result from invasion or compression by a tumor mass, often malignant, (c) occur secondary to renal disease itself, notably hypernephromas, or (d) occur spontaneously, especially among children. The incidence of renal vein thrombosis and the nephrotic syndrome at the Mayo Clinic was 0.6 per 1000 autopsies.[22] Constrictive pericarditis[24] and venous hypertension, at the right heart level,[6] can cause membranous glomerular changes and the nephrotic syndrome. The association of degenerative arterial disease with nephrosis is not widely appreciated.[25] Frequently there is a widespread nephrosclerosis; it can also be produced by localized disease. The nephrosis that in rare instances accompanies sickle cell disease[26] is probably related to the favorable milieu in the kidney for sickling progressing to thrombosis. Tubular damage and glomerular

engorgement with basement membrane thickening occurs. A normal serum cholesterol level is an atypical feature of this entity. A nephrotic syndrome that accompanied a pheochromocytoma regressed after removing the tumor.[27] It is likely that protracted vasoconstriction and ischemia may be the underlying mechanism. Renal tubular necrosis on a vasospastic basis has also been associated with pheochromocytomas.[28]

It is important to put the overall incidence of the various types of nephrotic syndromes in proper perspective. Their distribution in 358 cases, based upon renal histological diagnoses and clinical associations, is summarized on Table II. It can be seen that two-thirds of the patients remain dissociated in the "idiopathic" variety. Only diabetes and lupus occur with any degree of frequency among the other patients.

Table II

Nephrosis — Cause in 358 Cases

	Per Cent
Glomerulosclerosis	62.5
Diabetes M	15
Lupus	9
Amyloidosis	3
Nephrosclerosis	2
Renal Vein Hypertension	1.5
Miscellaneous	7

Proteinuria is the hallmark of the nephrotic syndrome. Autopsy protocols from 84 patients who had varying degrees of proteinuria prior to death were analyzed (Table III). A reason for the proteinuria was evident in all cases and 35 per cent had more than one possible mechanism present.

Table III

Causes for Proteinuria — (84 Autopsies)

	Per Cent
Single possible etiology	65
Multiple possible etiologies	35
Nephrosclerosis	27
Congestive heart failure	21
Fever (over 101°)	17
Diabetes (6 had Kimm. Wils. disease)	15
Pyelonephritis	11
Hydronephrosis	7
Chronic nephritis	4
Miscellaneous	30
Uremia (3 more without proteinuria)	14

Severe nephrosclerosis was the most frequent associated pathology. There was no relationship between the degree of proteinuria and the age of the patient when their nephrosclerosis occurred. Pathophysiological conditions such as fever and congestive heart failure were frequent causes. These observations are presented to emphasize the significance of proteinuria, no matter how mild it may be.

A complete set of references may be obtained by contacting the author.

II. Selected Clinical Aspects of the Nephrotic Syndrome

Capt. Timothy Harris*

When one thinks of the clinical picture of the nephrotic syndrome, the tragic vision commonly called to mind is of a phlegmatic moon-faced cushingoid child, pale and usually massively edematous. Indeed, on any active pediatric ward, one or more of these children may be seen during a year. The estimated incidence is two per 100,000 children. In the adult, the full blown clinical picture is usually not much different.

However, the nephrotic syndrome may run the gamut, from a completely asymptomatic patient, a patient bothered by an underlying collagen or infectious disease to the picture of massive renal and hormonal imbalance, discomfort and deformity. The laboratory findings and the pathogenesis of this disease have been eloquently covered, and indeed it is impossible at the present time to separate these aspects from the clinical picture of nephrosis. SLE, or other so called connective tissue or autoimmune disorders, bee stings and ingestions or congestive heart failure may be the presenting syndrome, and nephrosis may be diagnosable only by laboratory means or with a high index of clinical acumen.

Nephrosis or the clinical nephrotic syndrome has been known for centuries. It is of interest that some statues of the sick at the temple of Apollo in Delphi may represent the massive edema of the nephrotic syndrome.

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William of Salicet, a 13th century physician of Bologna, Italy, described the first association of renal disease and the nephrotic syndrome. This worthy remarked upon the association of dropsy, an anasarcous state, and nephritis.

In 1476, Saliceto of Italy made the association of "swelling of the legs and abdomen, and scanty urine with hardened kidneys."

A hiatus existed until the early 1800's when William Charles Wells, an esteemed London practitioner, demonstrated a material in the urine of patients with dropsy. This material coagulated with boiling. Some of the patients were known to have had an antecedent scarlatina infection, prior to their edematous state.

It remained for Dr. Richard Bright to establish the definitive clinical and laboratory correlation of proteinuria, dropsy, pathologic renal changes and scarlatina. The three cases originally described by Bright in 1823 have been recently reviewed by E. L. Becker, who has resectioned and examined the pathologic material in the light of present day methods. The etiologic factors were renal amyloidosis due to tuberculosis, nephrosclerosis and glomerulonephritis.

In 1942, Ellis attempted to correlate the clinical picture with the pathologic anatomy of the renal lesion. He described two pathologic entities.

The first type of lesion, proliferative glomerulonephritis or Ellis type I was associated with the following signs. Early, the symptoms were abrupt in onset with malaise, headache, fever and occasional back, flank or abdominal pain; 84 per cent of his cases had an antecedent infection, usually a sore throat; 90 per cent had clinical edema primarily of a moderate degree; the age range was variable but 60 per cent were under the age of 20. There was complete recovery in 82 per cent and death either acutely or chronically in 18 per cent.

Ellis type II nephritis pathologically was a membranous glomerulonephritis associated with an insidious clinical onset. A prior infectious incident was practically non-existent (5%); as the clinical picture progressed, edema was persistent and recovery was rare. It is important to note that infection was the most common problem and complication of this group and that the more familiar problems of chronic renal damage, e.g. hypertension and cardiac failure were encountered only in advanced stages of the disease.

These early observations kindled excitement and prognostic hope. However, the increase in knowledge through the years has made a clinical and pathologic correlation less exact. A whole host of renal lesions have now been demonstrated to produce the range of clinical signs and there has been extensive overlap between clinical picture and underlying pathologic change.

Clinically, the nephrotic syndrome has suffered some confusion of definition, for the classic Tetrad of proteinuria, hypoalbuminemia, hyperlipemia and oedema may in early cases be lacking. Berman has shown that many patients may present only with proteinuria and the doubly refractile fat bodies; the other parameters being met only as disease progressed.

The syndrome occurs most frequently in the younger age groups with the clinical and laboratory picture extending into the 80 year old range.

The etiology as has been discussed may run the gamut:

1. Streptococcal infections
2. Diabetes mellitus
3. Renal vein thrombosis
4. Amyloidosis, renal
5. Pulmonary or systemic tuberculosis
6. Carcinoma
7. Chronic infection
8. Rheumatoid arthritis
9. Hodgkins disease
10. Chronic ulcerative colitis
11. Autoimmune disorders
12. Drug ingestion
13. Bee stings and allergic responses, etc.

The clinical picture is usually one of insidious and protean progression — weakness, lassitude, syncope may be early signs followed by insidious weight gain preceding the development of clinical edema.

The fluid accumulation was originally associated with periorbital and leg edema, but fluid may accumulate in any body cavity, e.g.: pleura, abdomen, etc., to augment the patient's distress.

As the edema progresses, the patient will usually show signs of anorexia, nausea, diarrhea, irritability, etc. Calcium metabolism is affected

and a slowing of child growth is quite common. The duration of the clinical syndrome may be months or years.

The edema may be quite impressive and demoralizing. The psychiatric guidance of the depressed patient with poor reserve due to the debilitating disease may be a tremendous problem, for patients commonly tend to equate the severity of their disease with their physical deformity and the relief of nephrotic edema may be extremely difficult.

Infection continues to be the great problem in these patients and, indeed, prior to antibiotic therapy the Guy Hospital figures reveal intercurrent infection to be the most common cause of death in the bloated nephrotic.

Fortunately, the clinical course has been modified by the advent of antibiotics and steroids. It is still generally felt that children do better than adults and that with steroid therapy, childhood survival and remission have been markedly improved. Today, 75 per cent of affected children now reach the asymptomatic state.

The natural history of the disease is fascinating, dramatic, enigmatic, and challenging to the clinician, for the disease is one of fluctuations, remissions, cures, deaths and is quite unpredictable although infection has been handled more effectively.

Prognosis mainly depends on the underlying renal disease, as well as the etiologic agent. In those acute cases where steroids and support can tide over an acute renal insult the outlook is good; however, where the disease involves progressive renal failure the outlook becomes grim with the attendant cardiovascular and CNS problems — infection and uremia, the ultimate cause of death. Infection is less of a problem but takes its toll.

The diagnosis and management of the nephrotic syndrome still presents a great challenge to the clinician. The patient in his deformed state is a therapeutic and emotional problem and the evaluation and treatment of the underlying disease process may be both stimulating and rewarding.

(to be continued)

Second Biennial Seminar on Prematurity

The Arizona State Department of Health and the State Advisory Committee on Prematurity will sponsor the Second Biennial Seminar on Prematurity, February 25-26, 1967, at the Executive House Arizonian, in Scottsdale, Arizona.

Guest speakers will be Dr. Sydney Gellis, Boston, Professor of Pediatrics at Tufts University; Dr. Marshall Klaus, Palo Alto, California, Director of Clinical Center for Premature Infants at Stanford University; and Dr. William Silverman, New York, Professor of Pediatrics at Babies Hospital, Columbia Presbyterian Medical Center.

Dr. Gellis will speak on "The Responsibilities of the Obstetrician and the Pediatrician to the Premature Infant" and "Clues to Congenital Defects in Premature Infants"; Dr. Klaus' subjects will be "Current Physiologic Therapy for Hyaline Membrane Disease" and "Touch and No-Touch Mothers in the Premature Nursery"; and Dr. Silverman will talk on "Thermogensis in the Neonate" and "Fetal Undergrowth."

The registration fee will be $7.50, which includes the Saturday luncheon. The meeting is open to all physicians and nurses. Advance registration is encouraged. Further information may be obtained by writing to: Frederic W. Baum, M.D., Chief, Maternal & Child Health Section, Room 402, Goodrich Building, 14 North Central Avenue, Phoenix, Arizona 85004.

Announce Annual Scott and White Conference

The 15th Annual Scott and White Conference in Medicine and Surgery will be held in Temple, Texas, February 19-21, 1967, at the Elks Club, 2613 Airport Road.

Guest speakers for the scientific program will be Dr. Denton A. Cooley, Houston, Professor of Surgery at Baylor University College of Medicine, and Dr. S. Gilbert Blount, Jr., Denver, Professor of Medicine and Head of the Division of Cardiology at the University of Colorado Medical Center.

Mr. George Jessel, world-famous Hollywood entertainer, will be the after-dinner speaker.

Coming Meetings

First Annual Meeting of the Society for Cryo-Ophthalmology, Las Vegas, Nevada, The Dunes Hotel, January 8-10, 1967.

Southwest Allergy Forum, Galveston, Flagship Hotel, January 19-21, 1967.

American College of Surgeons, Arizona Chapter, Tucson, Arizona Inn, January 20-21, 1967.

New Mexico Medical Society's Conference of County Medical Society Officers, Albuquerque, Sheraton Western Skies, January 21, 1967.

19th Annual Midwinter Radiological Conference, Los Angeles Radiological Society, Los Angeles, International Hotel, January 28-29, 1967.

TMA District 1 Meeting, Pecos, February 4, 1967.

21st Annual Symposium on Fundamental Cancer Research, The University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, February 27-March 1, 1967.

Colorado Winter Clinics, Denver, Brown Palace Hotel, February 28-March 3, 1967.

American Academy of Pediatrics, San Francisco, Hilton Hotel, April 3-4, 1967.

American Society of Internal Medicine, San Francisco, St. Francis Hotel, April 7-9, 1967.

Arizona Chest Disease Symposium, Tucson, April 8-9, 17967.

Southwestern Surgical Congress, Phoenix, Del Webb's Townehouse, April 10-13, 1967.

American College of Physicians, San Francisco, Fairmont Hotel, April 10-14, 1967.

American Academy of Neurology, San Francisco, Hilton Hotel, April 24-29, 1967.

Arizona Medical Association, Phoenix, Townehouse, April 26-29, 1967.

American Cancer Society, Dallas, Sheraton-Dallas Hotel, May 3, 1967.

American Gynecological Society, Phoenix, Arizona Biltmore Hotel, May 4-6, 1967.

Texas Medical Association, Dallas, May 4-7, 1967.

85th Annual Meeting of the New Mexico Medical Society, Santa Fe, La Fonda Hotel, May 18-20, 1967.

48th Biennial Meeting of the Southwestern Medical Association, El Paso, Sheraton-El Paso Motor Inn, February 8-10, 1968.

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"'Tranquilizer' is not a good word"[1]

"THIS classification is psychologically too seductive, pharmacologically too unspecific, and in terms of results not infrequently untrue."[2]

What is a tranquilizer? According to the 24th Edition of Dorland's Medical Dictionary[3] a tranquilizer is "an agent which acts on the emotional state, quieting or calming the patient without affecting clarity of consciousness."

Defining a drug by its effects, however, can be misleading. The same effects by which the dictionary defines a tranquilizer have sometimes been seen after administration of a sedative — or, for that matter, a placebo.

Ambiguous though the term may be, it appears to be here to stay. The 1966 edition of the Physicians' Desk Reference[4] lists 42 tranquilizers indicated for treatment of anxiety and apprehensive states.

'Tranquilizers' have differences in action, differences in effect

Although all tranquilizers are intended to calm anxious patients there are differences in their actions — and in their effects. They have been divided into three categories — the rauwolfia group, the 'minor' tranquilizers, and the phenothiazines.[5]

Although the tranquilizing effect of rauwolfia has been known for centuries, its use as an antipsychotic agent in current practice has diminished.[5]

A 'minor' tranquilizer is often prescribed to achieve more than one effect. Thus, besides being tranquilizers some of these compounds may be muscle relaxants, antihistaminics with some calming action, anticholinergic sedatives, or antispasmodics.[5]

The phenothiazines are considered 'major' tranquilizers because they alter psychotic behavior.[1]

This classification may have done them more harm than good because it implies that the phenothiazines should be reserved for the more severely disturbed. This is not necessarily true.

The phenothiazines — and the problem of sedation

One of the problems of prescribing phenothiazines for ambulatory patients has been the fear that excessive sedation will impair the patient's ability to function. This, however, is less of a problem with some of the phenothiazines.

"Clinically they may be differentiated primarily in terms of their potency and the extent of their sedative effect, which appear to be inversely proportional. That is, the least potent, the one which is used in highest dosage, chlorpromazine, is the most sedative, while the reverse holds true for fluphenazine."[6]

In a recent report on various studies conducted over several years evaluating 360 patients treated for anxiety and stress with seven phenothiazines, this inverse relationship of potency to sedation was confirmed.[7] Also under consideration was the degree to which the particular phenothiazines neutralized anxiety (the angolytic index). Interestingly enough there was, again, an inverse relationship. The most sedative of the phenothiazines appeared to be the least active in neutralizing anxiety. Fluphenazine, one of the least sedative, on the other hand, was found to be most effective in relieving anxiety.[7]

RELATIVE SEDATIVE AND ANGOLYTIC INDICES OF PRINCIPAL PHENOTHIAZINES*

DRUG	SEDATIVE INDEX	ANGOLYTIC INDEX	BASED ON STANDARD DOSE OF
Chlorpromazine	100	15	25 mgs.
Triflupromazine	100	15	25 mgs.
Thioridazine	90	17	25 mgs.
Perphenazine	15	25	4 mgs.
Carphenazine	25	25	25 mgs.
Trifluoperazine	3.3	95	2.0 mgs.
Fluphenazine	3.5	100	2.5 mgs.

*adapted from Sainz[7]

Prolixin is therapeutically effective without excessive sedation

When used as a 'tranquilizer' in general medical practice, in many patients Prolixin (Squibb Fluphenazine Hydrochloride) suppresses anxiety, but not normal activity. These two features are of particular importance to patients who must be able to live and work without their normal daily activities being restricted.

Because of its longer duration of action, Prolixin, in doses of as little as one to three milligrams in adults, generally taken once a day, is effective in maintaining many patients free of their symptoms of anxiety and tension.

Contraindications: Do not use with high doses of hypnotics or in patients with subcortical brain damage. Use with caution in patients with a history of convulsive disorders. Severe reactions may occur in patients with idiosyncrasy to other centrally-acting drugs, and severe hypotension may occur in patients with special medical disorders, e.g. mitral insufficiency and pheochromocytoma.

Precautions: Effects of atropine, anesthetics and C.N.S. depressants may be potentiated. Hypotension may occur in patients undergoing surgery. Do not use epinephrine for treatment of the hypotensive reactions which may appear in patients on phenothiazine therapy.

Side Effects: Extrapyramidal reactions, allergic skin reactions, the possibility of anaphylaxis, drowsiness, visual blurring, dizziness, insomnia, nausea, anorexia, salivation, edema, perspiration, dry mouth, abnormal lactation, polyuria, hypotension, and jaundice and biliary stasis may occur. Routine blood counts are recommended to determine possible blood dyscrasias; if symptoms of upper respiratory infection occur, discontinue drug and institute appropriate therapy.

Available: 1 mg. tablets. Bottles of 50 and 500.

For full prescribing information, see package insert.

References: 1. Simpson, G.M.: Postgrad. Med. 39:557, 1966. 2. Freyhan, F.A.: Am. J. Psychiat. 115:577, 1959. 3. Dorland's Illustrated Medical Dictionary, ed. 24, Philadelphia, W. B. Saunders Co., 1965, p. 1603. 4. Physicians' Desk Reference, 1966, Oradell, N.J., 1965, p. 310. 5. Cohen, S.: Northwest Med.: 65:197, 1966. 6. Detre, T., and Jarecki, H.: Connecticut Med. 25:553, 1961. 7. Sainz, A.: Psychosomatics 5:167, 1964.

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